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CONTENTS

Some End Results of Fractures About the Elbow Joint. *James Warren Sever* 745
Treatment of Nasal Fractures. *L. W. Jessaman.* 753
Tuberculous Meningitis with an Unusual Cerebrospinal Fluid. *James W. Watts* and *Henry R. Viets* 757
Early American Experience Under the Coroner Laws. *Timothy Leary* 759
The Pathology of Diabetes in Young Adults. *Irvine H. Page* and *Shields Warren* 766
Sugar Tolerance and Hypertension in Cataract Patients. *E. R. Blaisdell* 768

MEDICAL PROGRESS

Progress in Pediatrics. *John Lovett Morse* 771

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Case 15151—Two Weeks' Cough and Vomiting. *Richard C. Cabot.* Case 15152—Three Days' Cough with Blood-Spitting. *Richard C. Cabot.* 779

EDITORIALS

Public Health Problems Before the Legislature. 785
Christmas Seals 786
Diagnosis by Optical Machines 786
A Committee on Relationships of Nurses and Physicians 786
This Week's Issue 786
Can This Be So? 787

(Contents continued on next page)

Medical Clinics for March

SOUTHERN INTERURBAN CLINICAL CLUB NUMBER

linic of Dr. C. C. Bass, New Orleans, La.
Pellagra.
linic of Dr. E. Bates Block, Atlanta, Ga.
A Case of Dystrophia Adiposogenitalis with Disappearance of the Testes.
linic of Dr. C. Sidney Burwell, Nashville, Tenn.
Three Types of Circulatory Failure.
The Interpretation of Changes in Body Weight.
linic of Dr. C. L. Eshleman, New Orleans, La.
Some Types of Hypertension.
linic of Dr. Bryce W. Fontaine, Memphis, Tenn.
A Case of Tropical Sprue Endemic in Tennessee.
linic of Dr. Lewis M. Gaines, Atlanta, Ga.
Diagnostic Problems in Organic Neurology.
linic of Dr. George Herrmann, New Orleans, La.
Cardiothoracic Distress (Types). Differentiation and Treatment with Especial Reference to Sympathectomy.
nic of Dr. W. R. Houston, Augusta, Ga.
The Spasmogenic Aptitude.
ic of Dr. Chaille Jamison, New Orleans, La.
A Case of Pernicious Anemia.
linic of Dr. F. M. Johns, New Orleans, La.
Amebiasis, with Special Reference to Its Treatment with Iodo-oxy-quinolin Sulphonic Acid.
linic of Dr. Isaac Ivan Lemann, New Orleans, La.
Osteitis Fibrosa Cystica in an Infantile.
Bronchial Polyp Associated with Mediastinal Mass (Probably Mediastinal Lymphnodes); the Latter Causing the Presenting Symptom of Dysphagia.
linic of Dr. Randolph Lyons, New Orleans, La.
Report of a Case of Arteriosclerosis, Myocarditis, Pulsus Alternans, and Possible Coronary Occlusion. Value of Certain of the Newer Diuretics in Cardiac Edema.
Clinic of Dr. Cabot Lull, Birmingham, Alabama.
Hemoptysis: Its Diagnostic Importance.
Clinic of Dr. James S. McLester, Birmingham, Alabama.
Thyroid Deficiency as a Cause of Poor Health.
Clinic of Dr. Hugh J. Morgan, Nashville, Tenn.
Induced Syphilitic Meningitis (Meningo-recurrences).
Clinic of Dr. John H. Musser, New Orleans, La.
The Leukocytic Response to Throat Infections.
Clinic of Drs. James E. Paullin and Harold M. Bowcock, Atlanta.
Glycosuria.
Clinic of Dr. Paul H. Ringer, Asheville, N. C.
The Problem of Collapse Therapy in Pulmonary Tuberculosis.
Clinic of Dr. Stewart R. Roberts, Atlanta, Ga.
Neuropathic Dropsy of the Right Forearm and Hand.
Acute Pellagra Associated with a Manic-depressive Psychosis Developing after a Five-year Pre-pellagrous Period.
Scleroderma Associated with Symptoms of Erythromelalgia and Myxedema.
Clinic of Dr. G. Canby Robinson, Nashville, Tenn.
A Case of Coronary Occlusion, Associated with Ventricular Paroxysmal Tachycardia.
Clinic of Dr. V. P. Sydenstricker, Augusta, Ga.
Wilson's Disease (Progressive Lenticular Degeneration).
Sickle-cell Anemia.
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CONTENTS

(Continued)

MASSACHUSETTS MEDICAL SOCIETY
Section of Obstetrics and Gynecology ... 787
The Treasurer's Report ... 788

BOOK REVIEWS
Angina Pectoris. Harlow Brooks ... 796
The Harvey Lectures. 1927-1928. ... 795
International Clinics ... 795
Spinal Anesthesia. Charles H. Evans ... 796
Surgical Clinics of North America, February, 1929, Vol. 9, No. 1 ... 796
A Textbook of Medicine ... 795
Tumors Arising from the Blood Vessels of the Brain. Harvey Cushing and Percival Bailey ... 795
You—and the Doctor. John B. Hawes, 2d ... 794

BOSTON MEDICAL LIBRARY
Trench Mouth ... 789

CORRESPONDENCE
Criticism of an Editorial. Frank E. Rowe ... 789
Suffolk District Medical Society. Arthur H. Crosbie ... 789

MISCELLANY
Why a Graduate Course in Cancer ... 770

NEWS ITEMS ... 792

NOTICES ... 792

NOTICES OF MEETINGS ... 792

OBITUARIES ... 790

RECENT DEATHS ... 790

SOCIETY MEETINGS ... 794

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The next Annual Meeting of the Massachusetts Medical Society will be held at the Copley-Plaza Hotel in Boston, June 10, 11 and 12, 1929

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VOLUME 200 APRIL 11, 1929 NUMBER 15

SOME END RESULTS OF FRACTURES ABOUT THE ELBOW JOINT*

BY JAMES WARREN SEVER, M.D., F.A.C.S.†

REAL end result studies are always of interest, and in this paper, I propose to report results of twelve cases of fractures about the elbow. Some of these cases have been seen as much as eight years after their original injury; some less than two years. They all represent fractures involving the elbow joint, and are mainly of the type known as supracondylar, with associated lesions of other types.

Some were reduced and some not, but in spite of that latter fact, the results are strikingly similar. This does not mean to imply, however, that in these cases of elbow fracture, no treatment, so far as an adequate reduction is possible, is necessary for a good result. They simply show, in so far as they individually are concerned, that they have worked out their own salvation, as well as excellent function, in spite of severe injuries.

Among the most interesting factors in this series are the adaptive bone changes as the result of time, growth and function. The x-rays taken at the time of the accident, and those taken several years later, seem to represent totally different conditions, and seemingly impossible adaptive changes.

Supracondylar Fractures: All but three of the following ten cases were in children. This condition of youth alone alters the whole prognosis. In children less violence is generally needed to produce a fracture than a similar condition in an adult. They naturally tend also to follow epiphyseal lines and generally do not lead, except when compound, to much damage of the soft parts. It is well known, however, that the supracondylar fracture is the one *par excellence* for the development of the much dreaded Volkmann's ischaemic paralysis. Therefore, early fixation in acute flexion should be done with due caution and great subsequent watchfulness.

The diagnosis of a supracondylar fracture is generally not difficult. The usual signs are present, such as swelling, generally severe, crepitus with marked and rather free lateral mobility of the lower arm and condyles of the humerus, which move as one. There is, of course, unless there is a complicating "T" fracture of the lower end of the humerus, no disturbance of the usual relation of the two condyles to each other. There is generally marked backward displacement of the elbow on the shaft of the humerus.

X-rays will show laterally the line of fracture with the generally marked backward displacement of the condyles. Not infrequently the lower fragment is still connected with the back of the humerus by a periosteal strip.

An anterior-posterior view should always be taken, before and after reduction better from

CASE 1; X-RAY NO. 1: June 6, 1914. Supracondylar fracture with imperfect reduction.

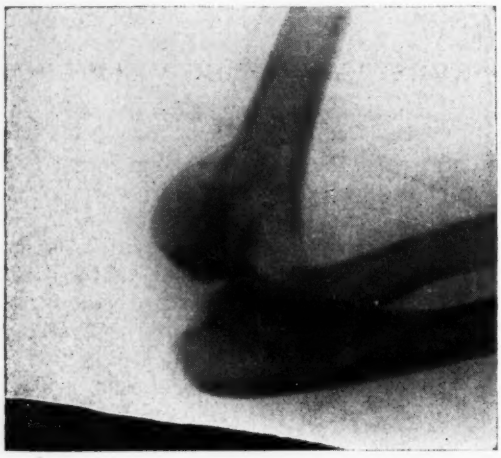
behind, with the elbow flexed. This will show whether or not the lateral displacement has been reduced. If this has not been accomplished, one must look forward to a diminished or reversed carrying angle at the elbow, due to this displacement in part and also to rotation of the shaft of the humerus.

Reduction is best attempted with the arm in abduction and outward rotation, so as to control these above mentioned factors. After reduction of the fragments, by manipulation, the arm may be placed in acute flexion unless the swelling, or interference of circulation by such a position contraindicates it. An internal angular splint may also be used, but personally, I prefer the acutely flexed elbow as the key position.

*Read before the Boston Orthopedic Club, November 13, 1928, and the Pentucket Medical Club, Haverhill, December 14, 1928.

†For record and address of author see "This Week's Issue," page 786.

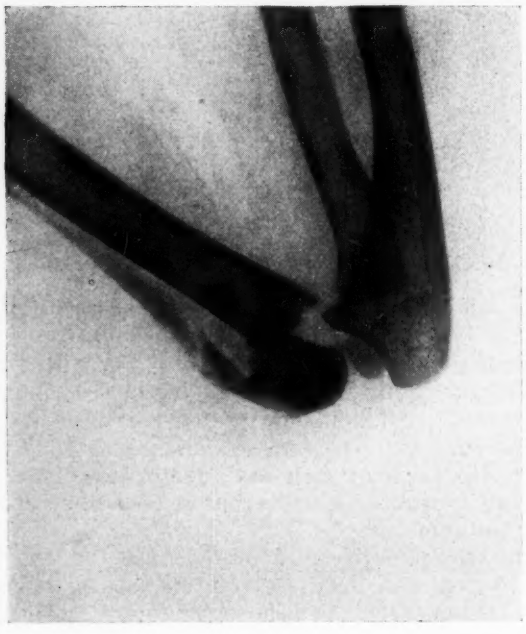
CASE 1, R. T., age 7 years: Accident June 6, 1914; fell off a tree; supracondylar fracture; internal angular splint. *X-ray no. 1*, shows position after reduction. Still some backward displacement of lower end of shaft, at epiphyseal line, with riding forward of proximal end of humerus.
Condition, March 15, 1922, age 14 years: Normal



CASE 2, X-RAY NO. 2: June 15, 1914. Supracondylar fracture before reduction.

motion in flexion and extension. Reversed carrying angle; considerable deformity of external condyle; supination and pronation normal. A good result which should not be expected from such a fracture incompletely reduced in two planes.

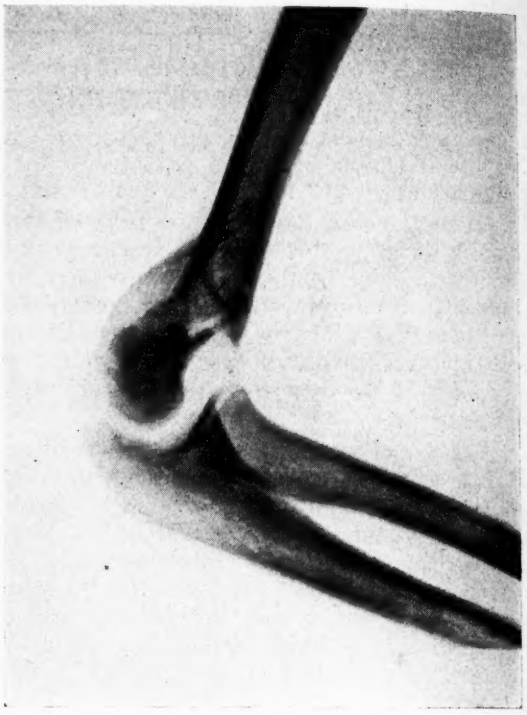
A reversed carrying angle is not a serious defect and really does not impair the function of the arm much. It is really largely cosmetic.



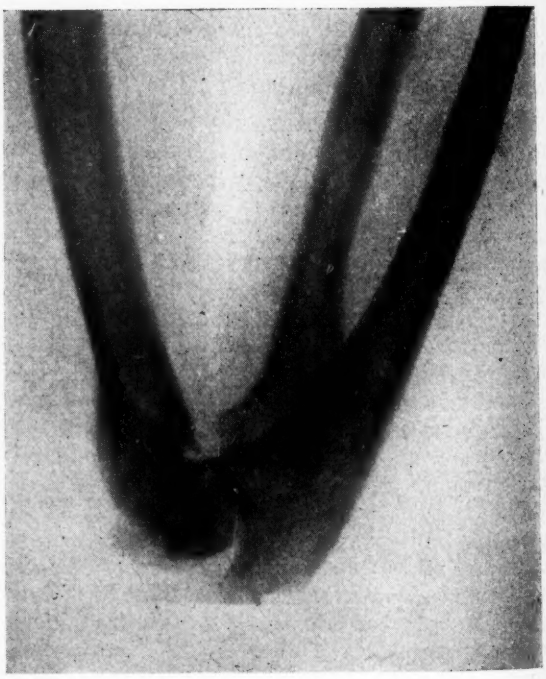
CASE 2, X-RAY NO. 3: Eighteen days after partial reduction; fragment still displaced backward, with periosteal bridge forming on back of humerus.

CASE 2, J. T., age 5 years: Fell down stairs June 14, 1914; supracondylar fracture of humerus; reduced under ether; acute flexion. See x-rays no. 2, before reduction; x-ray no. 3, 18 days after reduction; note, incomplete reduction of lower fragment with marked periosteal bridge forming at the back of the shaft of the humerus.

This boy was seen August 1914, two months after the accident, and was apparently discharged from



CASE 3, X-RAY NO. 4: August 14, 1916. Supracondylar fracture before reduction.



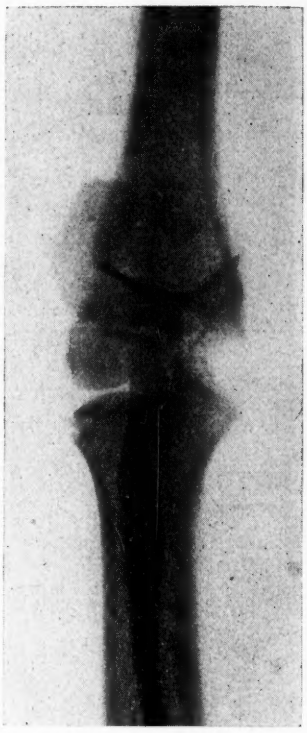
CASE 3, X-RAY NO. 5: Supracondylar fracture after reduction; arm in acute flexion.

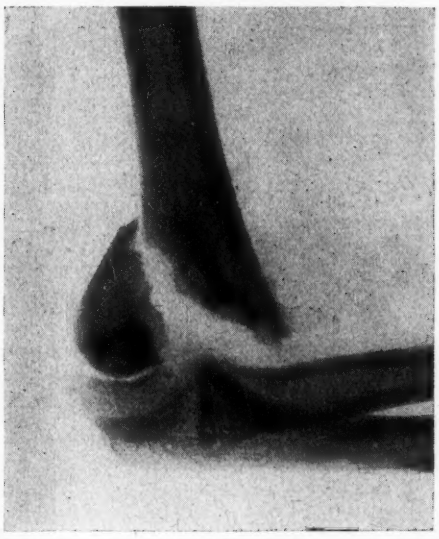
treatment at that time, with the note that he had a good result and good motion.

This case, although representing an incomplete reduction, evidently through adaptive growth, obtained a useful functioning joint. Further attempts, however, should have been made to reduce the fracture.

CASE 3. D. F., age 11 years: Accident August 14, 1916; fell on left elbow, causing a supracondylar fracture. (See x-ray no. 4 before reduction.) The fracture is a little higher than the usual supracondylar type and well above the epiphyseal line. There is moderate backward displacement of the lower distal fragments.

The second x-ray (no. 5) taken August 19, 1916, after reduction, shows excellent position. The elbow is in acute flexion.





CASE 4, X-RAYS NOS. 6 AND 7: Lateral and anteroposterior view of a supracondylar fracture, May, 1914, before reduction.

This boy was seen five and a half years later, March 1, 1922. At that time there was normal motion in all directions; all bony landmarks appeared normal, and the functional result was perfect. This is more or less what should be expected, following such a good reduction.

CASE 4, R. K., age 11 years: Accident, May 19, 1914; fell off a fence. The x-rays (See x-rays nos. 6 and 7) taken at the time, show a beveled fracture of the lower end of the humerus, with backward displacement of the elbow and a displacement downward of the proximal fragments. By pulling the elbow forward and acutely flexing, a good reduction was obtained. (See x-ray no. 8.)

This boy was seen February 15, 1922, age then 19 years. At that time there was normal motion in flexion. Extension, however, was limited to a little less than a right angle. Pronation and supination were normal. He was seen with the question of an operation to be performed in order to give him more extension. This was advised against, because he seemed to have a good useful arm, and it was felt that no promise could be made about increasing his range of motion.

X-rays (See x-ray no. 9) taken February 1922, show a normal elbow with the exception of the olecranon fossa, which is partially filled with callus, thus blocking the olecranon in extension.

CASE 5, D. D.: This boy appeared at the Hospital in October 1919, having fractured his right arm about a year previously. The x-ray (see x-ray no. 10) shows the result of a supracondylar fracture; a marked reversing of the carrying angle at the elbow, and a rotary displacement of the lower fragment. The function, except for the loss of the carrying angle, was perfect. By means of a supracondylar osteotomy, I attempted to restore the carrying angle, or at least partially restore it. In this there was some success, but not perfect.

The second x-rays show the condition in March 1922, over two years after the operation. (See x-ray no. 11.)

As in all these cases of reversed carrying angle with the elbow flexed, the condition is not suspected or noticed, but in extension there is a marked rotary movement at the elbow which throws it into valgus.

CASE 6, R. W., age 7 years: This boy fell and fractured the ulna and dislocated the head of the radius of the left arm August 14, 1914. He was treated with fixation by an internal angular splint for this condition.

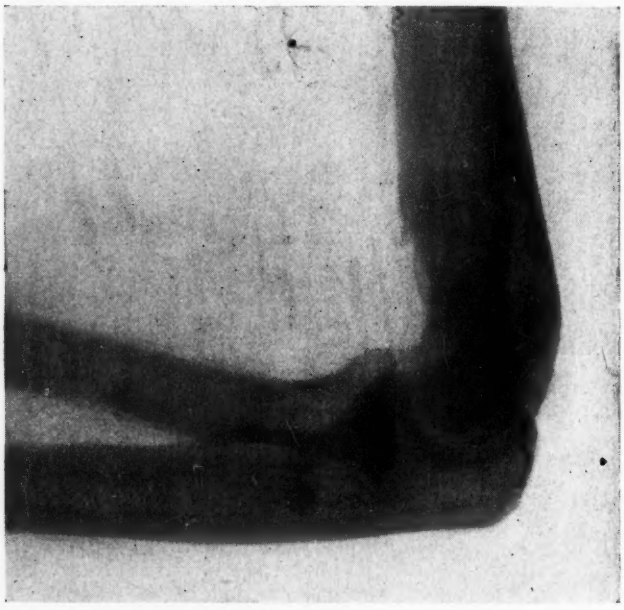
June 12, 1916, he fell again and suffered a supracondylar fracture of the same arm. The fracture was reduced in part only and the arm put in right angle flexion; this position was maintained several weeks, following which, the patient had some physiotherapy until August 1916. He was seen six years later, on March 15, 1922, on which date he showed perfect motion at the elbow without any deformity. Pronation and supination were normal, in spite of his previous accident to the head of the radius. He was then 13 years of age.

The x-ray (see x-ray no. 12) shows the condition of the fracture with an internal angular splint applied; there is the usual backward displacement of the distal fragment as well as a partial dislocation of the elbow joint.

CASE 7, D., age 45 years: Knocked down by an automobile November 11, 1924. This accident resulted in a fracture of the skull, multiple bruises, and a "T" fracture of the left elbow. The x-ray, (see x-ray no. 13) shows the line of fracture extending into the elbow joint, with a splitting off of the internal condyle. An attempt was made to readjust the fragments by traction and lateral pressure, followed by the application of an internal angular splint. This was worn about four weeks, followed by many weeks of physiotherapy.

The result, two years later, showed 25° of flexion beyond a right angle; 25° extension beyond a right angle; and supination only one half normal. Whether it would have been better, from the point of function, to have operated on this elbow and adjusted the fragments to a better anatomical position, I do not know. The result, while not wholly bad, is not good and promises no further improvement.

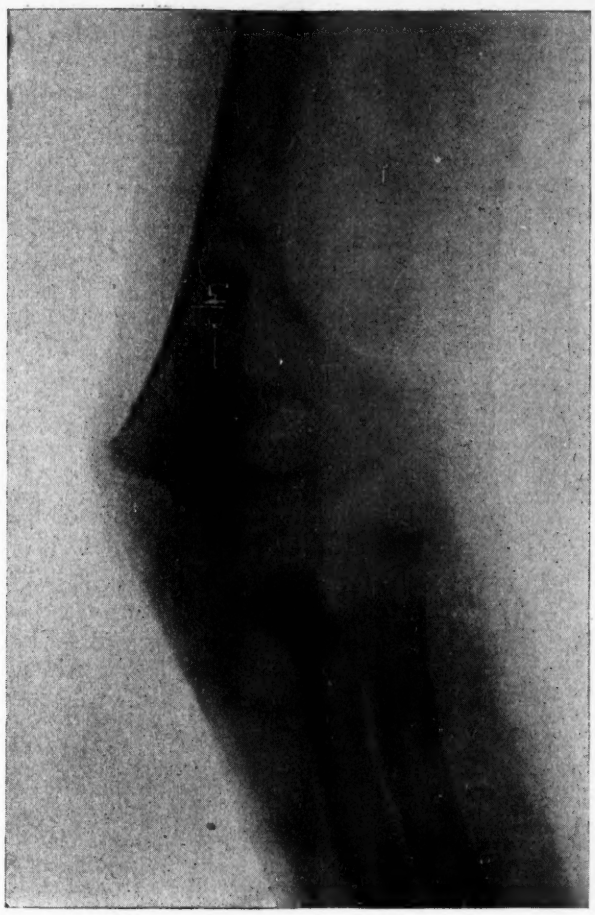
CASE 8, J. T., boy age 12 years: Fractured left elbow at the age of four. He had had no treatment at the time of the fracture. He was seen on the 7th of July 1916, eight years after the accident. The condition clinically showed marked increase in the carrying angle at the elbow. Flexion and extension were normal, as was also pronation and supination. No treatment or operation was advised. The x-rays at that time, 1916, showed a fracture of the external condyle of the humerus, with rotation of the fragment forward, including the capitellum. (See x-ray nos. 14-15.) There was also an elongation of the internal epicondyle. The boy evidently made an excellent functional recovery, without any treatment, from a bad fracture.



CASE 4, X-RAYS NOS. 8 AND 9.

NO. 8: Shows condition after reduction with arm in acute flexion.

NO. 9: Shows condition eight years later.



CASE 5, X-RAY NO. 10: Old supracondylar fracture with lateral displacement of condyles and loss of carrying angle.

CASE 5, X-RAY NO. 11: Two and a half years later following attempted correction of deformity.

CASE 6, X-RAY NO. 12, June, 1916: Supracondylar fracture not reduced.

CASE 7, X-RAY NO. 13, November, 1924: "T" fracture of lower end of humerus.

CASE 8, X-RAYS NOS. 14 AND 15, July, 1916: Represents an old fracture of the elbow of eight years duration. Normal motion.

CASE 9, D. C., boy aged nine years: Fell from a tree April 5, 1919, and sustained a fracture of the external condyle of the left humerus as well as a

CASE 9, X-RAYS NOS. 16 AND 17, April, 1919: Antero-posterior and lateral views of elbow, showing fracture of the external condyle and upper end of ulna.

fracture of the upper end of the ulna, with slight displacement of the fragments.

He was seen three years later, February 15, 1922, age 12 years, at which time he showed normal extension and flexion at the elbow. Pronation and supination were normal. There was slight thickening about the external condyle. The functional result was perfect. This fracture was not quite high enough on the humerus to cause the not infrequent lateral displacement, leading to an increase in the carrying angle, so frequently seen.

The x-rays show clearly the anterior-posterior and lateral views taken the day of the injury. (See x-rays nos. 16 and 17.)

CASE 10, D. W., age 8 years, male: Fell and fractured the left elbow Dec. 12, 1914. This injury resulted in a fracture of the external condyle of the humerus, with moderate outward displacement and rotation forward of the fragments. He was treated by acute flexion, but no evident reduction of the fragments, which could have been reduced only by open operation.

The x-rays shown were taken March 3, 1915; at that time, he evidently had union but poor position. (See x-rays nos. 18 and 19.) He should also have had considerable limitation of flexion at the elbow from the blocking effect of the displaced fragment.

He was seen April 5, 1922, at which time, seven and a half years after the fracture, he showed practically normal flexion and full extension at the elbow. There was considerable widening at the joint, with slight decrease in the carrying angle. Supination and pronation were normal. This again is an excellent result of a practically untreated and unreduced fracture. Normal function was not interfered with in any way.

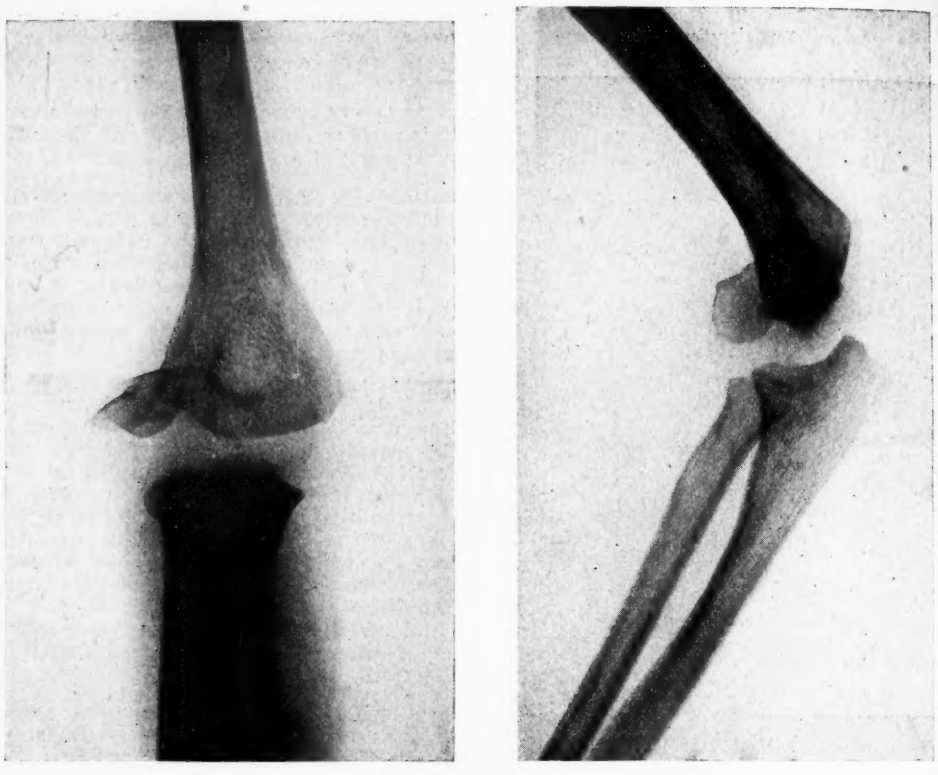
CASE 11, T. C., male, age 58 years: Fractured left elbow as the result of a fall on January 11, 1918. He sustained a comminuted fracture of the lower end of the humerus, which eventually resulted in considerable new bone formation on the anterior surface. This resulted in considerable interference with normal motion. He was treated by fixation in an internal angular splint only. He was seen June 26, 1919. The condition at that time was as follows: Flexes the elbow to just beyond a right angle and within 15° of full extension. Pronation and supination are normal. He has been seen many times since then up to within a year, and there has been no change and no increase in motion. He has, however, a fair functional use of the arm. Even at his age, an attempt to remodel the lower end of the humerus might result in better function. (See x-ray no. 20.)

CASE 12, J. L., male, age 29 years: This case is rather a different type and represents a complete fracture of the olecranon. This type of fracture naturally involves the elbow joint, but does not lead to much, if any, later disability.

Treatment may be by means of a straight splint with the arm fully extended, the fragments being held in place by cross strapping similar in type to that employed in fractures of the patella. Recently, the flexed position of the elbow has been advocated by some, combined with the use of pegs or screws to approximate the fragments.

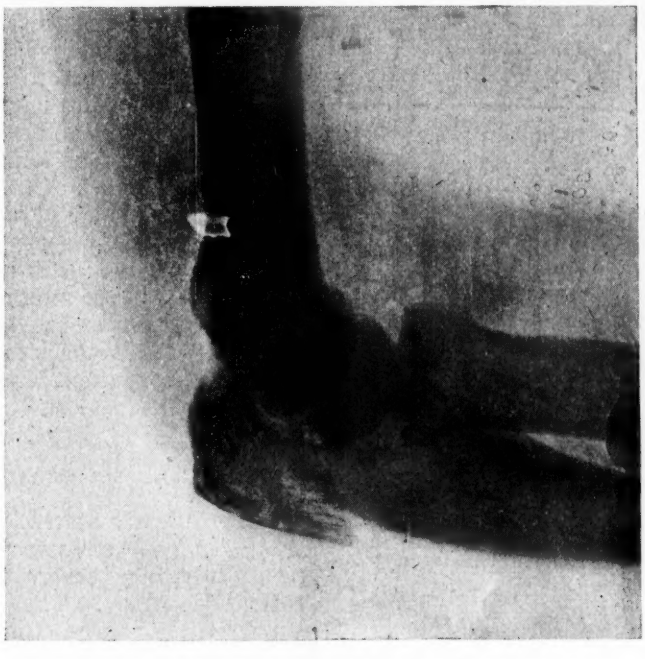
The fracture occurred Sept. 2, 1916, (see x-ray no. 21). The splint was off by October 7, 1916, and by December 1916, (see x-ray no. 22) there was active flexion to beyond a right angle and full extension. Supination and pronation, of course, were normal.

In these cases with much tendency to separation of the fragments, operative procedure may be justified to prevent, first of all, contracture of the triceps and later stretching of the fibrous union which occurs as the result of inadequate bony approximation, as the result of functional use. Bony union probably may not occur unless there is absolute approximation of the bone



CASE 10, X-RAYS NOS. 18 AND 19, March, 1915: Fracture of external condyle of humerus; normal motion without treatment seven and a half years later.

CASE 11, X-RAY NO. 20, September, 1918: Old fracture of the lower end of the humerus with exuberant callus.



CASE 12, X-RAYS NOS. 21 AND 22: Fracture of olecranon before and after union.

fragments. With such a bony union, an excellent functional result may be obtained. With much stretching of a fibrous union there is, of course, a moderate loss of extensor power.

TREATMENT OF NASAL FRACTURES

BY L. W. JESSAMAN, M.D., F.A.C.S.*

DURING late years there has been a great deal of work done relative to methods of correction in the different types of nasal deformity, including those due to some previous fracture. The results achieved in this work have been very gratifying. On the other hand, there is not the same amount of information available in regard to the proper treatment of recent nasal fractures. In looking over text books on the nose and throat it is surprising to find what very slight attention is given to this subject and how varied and usually inadequate is the infor-

*For record and address of author see "This Week's Issue," page 786.

mation given, in the main. Perhaps the reason for this condition of affairs is due to the fact that such a large percentage of these cases are treated by the general practitioner or the general surgeon and it is not until sometime afterward when the bones have become fixed in their new position and a definite deformity has resulted, (which in the majority of cases need not have occurred if the patient had received proper treatment at the time of injury), that the patient comes into the hands of the specialist. This may account for the fact that so little attention is given to the subject in works devoted to the nose and throat. A fracture of a leg or an arm is given painstaking care, but the nose which has been injured and displaced receives only passing regard and is considered of little importance.

Later, however, the patient becomes concerned over this facial deformity and seeks advice as to what can be done for its correction. These are the cases which at last come to the specialist for relief of a condition which in a large percentage of cases never would have existed had the original injury been properly cared for.

What has just been said is not a criticism of the individual doctor who may be called upon to treat a case of fractured nose, because, due to a lack of available information, he is not properly informed. It seems to the writer that our medical authorities, when writing text books, should give this subject more attention, and a proper description of the diagnosis and treatment which would be efficient and practical and yet not too complicated to carry out. It is, of course, true that if one reads through a sufficient amount of literature, he will be able to arrive at a fairly good working knowledge, but more readily available information is urgently needed.

Nearly all cases of fractured nose accompanied by displacement can be restored to the former position and shape if the case is properly handled soon after the injury. If, however, the bones are allowed to unite in their displaced position, the difficulties of treatment are very much increased. Then it may be very hard, if not impossible, to restore the nose exactly to its original shape.

ETIOLOGY

The cause of nasal fracture usually is the application of direct violence in some form. The applied force and its direction very largely determine the extent of the injury and the amount of deformity produced. There may be simply a disarticulation of the nasal bones and displacement slightly to one side; there may be a simple comminuted or a compound facture. If the force is in the right direction a depressed fracture may result.

DIAGNOSIS

In the ordinary case diagnosis is usually not difficult as the bones are found to be more or less movable and there is some displacement. Frequently, however, the physician does not see the case until marked swelling has occurred, concealing the deformity and making it more difficult to determine the presence or absence of fracture by the ordinary means of palpation. Usually, however, with a little patience and manipulation it is possible to determine displacement and crepitus even in the presence of pronounced swelling. If the skin is broken, the bones may be felt with a probe, and if the fracture is comminuted, the fragments are found to be movable. Bleeding from the nose usually occurs; this may be slight or profuse, but by the time the patient is seen by the physician it may have ceased. Examination of the nares, however, by means of a probe will frequently reveal a tear through the mucous membrane of the outer wall and the nasal process of the superior maxilla may readily be felt. The x-ray may be of assistance but frequently is without value.

PROGNOSIS

Prognosis, in the average case, is good as far as restoration of the nose to its former position is concerned, provided corrective treatment is instituted early, before there has been a union of the fragments in their new position. By their early replacement and maintenance of this position by means of pressure and correct splinting for a proper length of time, nearly all deformity can be prevented. If the nasal septum is displaced, it should be replaced at the same time treatment of the external deformity is carried out. In cases seen later it is often very difficult to restore the nose exactly to its former shape; therefore too much assurance should not be given to the patient and relatives, although a fairly good result can be obtained in nearly all cases.

TREATMENT

As previously stated, the question of proper treatment has been very much neglected. Treatment will vary somewhat with the type and extent of injury. The bone fragments should be replaced as early as possible after the injury. In the early cases this is fairly easy to accomplish, by the use of an elevator within the nose and external manipulation. In some cases proper forceps are extremely valuable. The writer has an Asch septum forceps which has been found very useful in fresh fractures and also in those of longer duration. This forceps is heavily made so that it is possible to refracture the nose, if necessary, when the bone becomes set in an abnormal position. One blade of Asch forcep can be passed into each nares and used to elevate the fragments. A blunt nasal elevator is needed, an ordinary nasal speculum, a medium length Killian speculum, and a good nasal forceps. These are the more essential instruments; others, of course, can be added

to meet the needs of the individual case or the preference of the operator. If it happens to be a case of compound fracture, the external wounds, of course, should be thoroughly cleaned and sutured if necessary. Care should be taken, however, not to remove bone fragments unless it is clearly evident that they have been torn from the periosteum to the extent that they are likely to slough out.

This attempt to preserve bone should not be carried to the point of leaving fragments when there can be little hope of their remaining. If it is a case where there is simply a disarticulation of the nasal bones and pushing to one side, all that may be required is to push them back into their former position, where they may remain. It is not best to do this and dismiss the patient, telling him that he will be all right, as the bones may be displaced again before union has occurred. An example of this injury is well illustrated in the following case:

CASE REPORT

S. E., aged 30, came to my office with the history that about one hour previously, while boxing he received a hard blow on the left side of the nose and after this found that the nose was displaced. Examination showed a rather marked displacement of nasal bones. With a moderate degree of force it was possible to push the bones back into normal position without an anesthetic. The nose then appeared to be so firm that no dressing was applied, but the patient was instructed to return the following day for observation which he did. The patient reported three or four times, as directed, at the office. The bones remained in perfect position and were soon firmly united.

This type of case, treated early, is very easily handled, but if allowed to go untreated for a few days, the bones become so firmly fixed that it is then difficult to replace them and there is also a greater tendency for them to slip out of place. Treatment then may require a general anesthesia and the use of instruments to replace the bones, packing in the nose and an external splint.

Other types of fractures constitute the majority and should have support over a period of time, this period depending on the extent of the fracture and the deformity. In some of these cases the bones can be replaced without an anesthetic, others require general anesthesia in order to lift the bone fragments into position. They should be held there by necessary support inside the nares in the way of packing and also an external splint which will bring pressure in the desired place. The packing in the nares should be placed high up and anteriorly. It need not be very large and it is not necessary in most cases to completely pack each nostril, as I have found that the packing placed high up remains in place remarkably well without much support from below. After having lifted the bones into position and placed the packing in the nares, the nose can be molded into its normal shape externally with the fingers and then a suitable external splint applied.

Any case having a fracture and displacement of bones of the nose is better protected if there is some form of external splinting, except in such instances as the case stated above, which did not require this support. The pressure should be at the sides and under no circumstances should pressure be exerted downward on the bridge of the nose as this tends to depress the fragments. The question, of course, arises as to what can best be used for this purpose. The application of a small piece of roller bandage on each side of the nose, held in position by bandaging, has been advocated. Plaster casts have been made, which, in turn, must be held in position and must be rearranged as swelling of the nose decreases. Modeling wax has also been used instead of plaster; also somewhat more elaborate apparatus has been arranged with a head band; another, which is attached to the teeth has fork-like prongs extending upward, to be placed in the nares for support. These are all good in certain cases, but what is urgently needed is a method which is fairly simple, and material that is readily available, and not too expensive.

A method of splinting which the writer has employed for a period of more than ten years is that of using strap iron about three-fourths inches wide and somewhat less than one-sixteenth inch thick and of sufficient length to mold about the nose and over the cheek. The strap iron is really a heavy hoop iron, readily bent with forceps and yet having a sufficient resistance to exert all the pressure that may be needed at the side of the nose; this pressure may be placed wherever required. Because of the difference in the size and shape of faces it is necessary to fit the splint in each individual case.

PREPARATION OF SPLINT

Having a piece of strap iron of the length estimated to be proper, as required in the individual case, cover it with adhesive tape so that no metal will come in contact with the skin. Using a pair of ordinary pliers, bend the splint to fit over the nose and over the cheek. The bend over the nose is always made high enough so that no pressure will be exerted downward and so depress the bones, the object being to have all pressure exerted at the sides. It may be necessary to use pressure on both sides, or perhaps only on the side toward which the bones were dislocated.

After having shaped the splint to conform to the face and having padded the part which goes over the nose to bring pressure in the proper location, long strips of adhesive are then attached to each end and by these the splint is held tightly down on the face. These strips of adhesive go completely around the back of the neck just below the auricle. At the apex of the bend over the nose another strip of adhesive is attached. This is long enough to extend well up over the forehead and is firmly held in

place by a cross piece of tape extending well to each side so that sufficient pull is maintained to keep it at the proper height. Figure 1 shows splint ready for use; figure 2 shows splint in position.

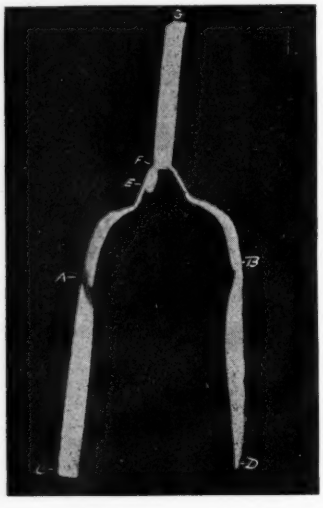


FIGURE 1.

The above figure represents one of the splints after it has been bent to fit the shape of the face in an actual case and was prepared ready for application. A and B represent the end of the splint. A to C and B to D are pieces of adhesive tape which are to be carried below each auricle and around back of the neck, thus holding splint firmly on the face. F to G is a strip of adhesive over the apex of the bend in splint which is to extend up onto the forehead to hold the splint at the proper height. E is a small pad of gauze fastened to the splint with adhesive tape to bring pressure to bear on the location where needed. In this particular case the nose had been dislocated to the right side and pressure was required on the same side to keep it from becoming dislocated again.

This external splint, when properly fitted to the face and pressure correctly applied to the nose, very rarely causes any particular discomfort. If, by any chance, there should be undue pressure on any particular point, it is very readily adjusted without disturbing the patient.

FIGURE 2.

The above figure shows a splint in position on the patient.

The splint is ordinarily allowed to remain in place for at least a week, although in some instances the bones may stay in position well enough to omit it sooner and in other cases a period of ten days may be required. The patient should be carefully watched, as it may be found necessary to rearrange a point of pressure over the nose. This is especially true if there is swelling at the time of its application, because as the swelling decreases, the effect of the splint is lost to a certain degree.

This method of splinting has given excellent satisfaction in my hands. The splint is readily made, can be adjusted to any face and pressure produced at any point desired. It also acts as protection against displacement of the bones by the patient as a result of accidentally hitting the nose after they have been replaced.

CASE REPORT

A. D., aged twelve: *History:* While wrestling with another boy about the same age, the patient fell, striking his nose against the corner of a table and on examination, his mother discovered that the nose seemed to be pushed to one side. Some epistaxis occurred. The patient was seen in the hospital about one hour after the accident.

Examination: The nose was pushed to the right side; there was some swelling and some bleeding. On palpation the nasal bones were found to be movable and apparently comminuted. The fracture apparently involved the nasal process of the superior maxilla.

Treatment: Ether anesthesia. With instruments the bones were replaced in position and then supported by intranasal packing. The nose was externally molded into the normal position and an external splint, such as has been described, applied. As the swelling began to decrease, the pressure was readjusted. A splint was kept on for about a week and when omitted the nose seemed to be fairly firm and as straight as it was prior to the injury.

Numerous case records might be included, but they would not add to the clearness of the subject and would prolong this article unduly.

SUMMARY

1. Information regarding fracture of the nose as given in our text books is, on the whole, very inadequate.
2. All cases of fractured nose should be treated as early as possible, as it is usually comparatively easy to replace it in normal position and obtain an excellent result unless the bones have been too badly comminuted.
3. After a fractured nose has become united in a dislocated position, it is very difficult often to replace it in its former position and obtain a satisfactory appearance.
4. Proper support is essential to hold the bone fragments in position until they have become reunited.
5. The use of strap iron, which is easily adjusted to the face, as described in this article, is extremely useful in handling these cases, as it forms an excellent external splint.
6. The splint needs to be readjusted when swelling has decreased. This can be done without disturbing the patient.
7. If the doctor who first sees the patient is not qualified to handle such conditions, he should call on someone who is competent to care for such cases to assist him.

TUBERCULOUS MENINGITIS WITH AN UNUSUAL CEREBROSPINAL FLUID

A Case Report*

BY JAMES W. WATTS, M.D.,† AND HENRY R. VIETS, M.D.†

THE case here reported is one of tuberculous meningitis, proved by guinea pig inoculation. Cerebrospinal fluid, obtained at the beginning of the disease, was under normal pressure and contained only 4 cells per c.mm. Cerebrospinal fluid examined six days later, however, showed the usual findings of tuberculous meningitis.

History: The patient, a single white man, 24 years old, was admitted to the Massachusetts General Hospital, July 7, 1928, complaining of headache and vomiting. His family history was significant; his mother, father, sister and uncle had died of tuberculosis when he was a child. His *past history* was unimportant. His *present illness* started two weeks before admission, when he began to have a generalized headache, not incapacitating. Ten days later he suddenly became dizzy and vomited. Vomiting, which was not projectile in character, continued four or five times a day until admission to the hospital; his headache became constant and severe.

Physical Examination showed a well developed, well nourished man, lying quietly in bed with eyes closed, complaining of nausea. His face was slightly flushed and his throat was reddened about the fauces. No abnormal breath sounds or râles in the lungs could be heard. B. P. 110/60; temperature 98.8°; pulse 50; respirations 20.

Neurological Examination: The patient was intelligent and clear mentally. There was no aphasia or apraxia. His sight was good; the right optic disk margins were blurred, but there was no papilledema; the veins were slightly engorged; the left fundus was normal. The other cranial nerves were normal. Co-ördination was good; no sensory disturbances were present; the deep reflexes were equal; the abdominal reflexes were absent. Plantar flexion was present on the right; no response on the left. There was no stiff neck or Kernig sign. His urine was normal. Stereoscopic roentgenograms of the skull showed the sella turcica a little larger than usual but within normal limits.

Course in the Hospital: July 7, 1928, the first lumbar puncture was done: the initial pressure was 150 mm. of water; on jugular compression the pressure rose to 240 mm. and fell promptly to 130 mm. on release. Cell count, 4 lymphocytes per c.mm.; total protein 83 mg. per 100 c.c.; Wassermann reaction negative; colloidal gold curve 0112100000. The temperature remained normal until July 13; his pulse rate ranged between 45 and 70. On July 11, the pupils were found to be unequal, his speech slow, with marked pauses after his words; later the same day he developed a partial aphasia. On July 12, he became irrational; his speech was not understandable. He took off his shirt and attempted to get out of bed. Lumbar puncture was done July 13. The initial pressure was 250 mm. of water; total protein 85 mg. per 100 c.c.; 205 lymphocytes per c.mm. The temperature rose sharply during the day to 101.8°; the pulse rate remained at 50. The leukocyte count in the blood was 11,500 per c.mm. The patient became drowsy and did not appear to understand what was said to him; the aphasia was more marked. The left side of the face and body became weak. The tone of the left arm and leg was increased; sensation was diminished generally and the left corneal reflex was obtained with difficulty.

On July 14, he became more lethargic. Cerebrospinal fluid examination revealed an initial pressure of 150 mm.; total protein was increased; 39 lymphocytes and one polynuclear leukocyte per c.mm.; sugar, 30 mg. per 100 c.c.; chlorides 605 mg. per 100 c.c.; Wassermann reaction, negative. A part of the cerebrospinal fluid was injected into a guinea pig and at autopsy, August 30, six weeks later, well marked tuberculosis was found. The next day the temperature was normal and the patient was rational. The pulse rate was 60. On July 16, the temperature rose to 100.5° and he appeared drowsy and irrational; pulse rate still 60. On July 17, his neck was stiff and the patient was partially comatose. The initial pressure of his cerebrospinal fluid was 200 mm.; 110 lymphocytes per c.mm.; total protein 87 mg. per 100 c.c.; sugar, 20 mg. per 100 c.c.; chlorides 611 mg. per 100 c.c.; colloidal gold curve 0112110000. The leukocyte count was 17,000 per c.mm. From this time until death, which occurred July 22, the patient remained in a semi-comatose condition. On July 19, cerebrospinal fluid examination revealed an initial pressure of 350 mm.; 227 cells per c.mm., 50% lymphocytes, 50% large round cells; protein 85 mg. per 100 c.c.; sugar, 20 mg. per 10 c.c.; chlorides 646 mg. per 100 c.c.; colloidal gold curve 1122220000.

A COMPARISON OF THE CASE REPORTED ABOVE WITH THOSE REPORTED IN THE RECENT LITERATURE

1. *Individual Cases*

Whitaker[8] reports the case of a patient entering the hospital for headache, dizziness, nausea, and vomiting. The temperature was normal, reaching 100° only twice in the first 6 days. The cerebrospinal fluid showed 40 lymphocytes; the globulin was not increased. There was a bilateral choked disk of 3½ diopters. The patient was operated for brain tumor; nothing was found. Autopsy revealed generalized miliary tuberculosis with meningitis. In our case the patient complained of headache, dizziness and vomiting; the temperature was normal 6 days; the right optic disk margins were blurred; cell count on the cerebrospinal fluid was normal. Our diagnosis was, as in Whitaker's case, brain tumor until a lumbar puncture was repeated 6 days after admission, following the development of new symptoms.

Benton[1] describes a patient first seen with a temperature of 99.6° and a pulse rate of 55. Lumbar puncture yielded fluid under diminished pressure; no cell count was done. A second puncture showed increased pressure and a lymphocytosis. This case was similar to ours in having a slow pulse, normal cerebrospinal fluid pressure, and a relatively normal temperature.

In a case reported by Wright[9] lumbar puncture done 3 days after the onset of the disease

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with convulsions showed the sugar 0.88% (not reduced), Pandy's reaction negative, and 100 cells, mostly mononuclears. Later the sugar was definitely reduced. This is of interest because the spinal fluid sugar was normal several days after the acute onset of the disease. In our case the sugar was not done early; when examined, on the 7th day after admission, it was reduced.

2. *Series of Cases*

1. *Pressure:* All authors report an increase of intraspinal pressure in tuberculous meningitis, but most of them give no manometric readings of the pressures. *The Human Cerebrospinal Fluid*[10] states that the pressure is considerably increased in nearly all cases and ranges from 300 to 700 mm. of water. Our case is unusual in that lumbar puncture was done in an early stage of the disease while the pressure was still normal; subsequent punctures gave increased manometric pressure readings.

2. *Cells:* In reviewing the literature from 1916 to June 1928, one finds reports that the cells per c.mm. in tuberculous meningitis vary considerably. In a series reported by Stewart[13] they range from 42 to 584 per c.mm.; by Greenfield and Carmichael[4] from 20 to 1000, averaging 100 to 300; in Kinnear's[5] series they average 73, but he does not give the upper and lower limits. Fontecella and Sepulveda[3] report counts ranging from 8 to 1000. Bickel[2] found the average count to be 230 cells, with a minimum of 0 and a maximum of 1400, but does not state the number of counts which were normal. In the series reported in *The Human Cerebrospinal Fluid*[10] the average count was 112, with a minimum of 0 and a maximum of 1000; five of the fluids had normal counts. McClelland[11] found cell counts ranging from 3 to 1870, with an average of 100 to 400, but he also did not state the number of normal counts. Reux[12] reports 3 cases with from 5 to 6 cells per c.mm. Our case, on the first lumbar puncture, showed 4 cells, practically a normal cell count. Cerebrospinal fluid examined from a puncture done six days later gave a count of 205 lymphocytes. This brings out two points: first, the clinical signs may be present for some time before the cells increase in the cerebrospinal fluid; second, the fluid may change rapidly in a few days from one which is almost normal to one characteristic of the disease.

CEREBROSPINAL FLUID EXAMINATION FROM LUMBAR PUNCTURE

DATE	INITIAL PRESSURE	CHARACTER	CELLS	PROTEIN	SUGAR	CHLORIDES	COLLOIDAL GOLD CURVE
July 7	150 mm.	Clear	4 Lymphocytes	83 mg.			0112100000
July 13	250 mm.	Clear Pellicle formed over-night	205 Lymphocytes	85 mg.			
July 14	150 mm.	Clear	39 Lymphocytes and 1 Polynuclear Leukocyte	Increased to alcohol & ammonium sulphate	30 mg.	605 mg.	
July 17	200 mm.	Clear	110 Lymphocytes	87 mg.	20 mg.	611 mg.	0112110000
July 19	350 mm.	Clear	227 Cells, 50% large round cells, and 50% Lymphocytes	85 mg.	20 mg.	646 mg.	1122220000

3. *Protein:* All authors report an increase of the protein in a majority of their cases: McClelland[12] found an increase in 92% and Stewart[13] in 83%. In 34 fluids reported in *The Human Cerebrospinal Fluid*[10] the average protein was 264 mg. per 100 c.c. On the first lumbar puncture in the case reported above, the protein was increased to 83 mg. per 100 c.c., the only abnormal finding in the fluid. This should have aroused our suspicions as the protein in uncomplicated brain tumor is not often so high. The protein ranged between 83 and 87 throughout the disease.

4. *Sugar:* The sugar content of the fluid shows a change soon after the distinct clinical symptoms of the disease are established and varies between 11 and 31 mg. per 100 c.c. (Greenfield and Carmichael[4]). *The Human Cerebrospinal Fluid*[10] records the average as being 27 mg. per 100 c.c. The sugar in our case was done for the first time 7 days after admis-

sion; at that time it was 30 mg. per 100 c.c. It fell to 20 mg. per 100 c.c. and remained at this level.

5. *Chlorides:* The chlorides are progressively reduced, as shown by Mestrezat[6] and Greenfield and Carmichael[4], and readings below 600 mg. per 100 c.c. are very suggestive of this disease. In Bickel's series[2], 25% of his cases gave readings below 610 mg. per 100 c.c.; in Norwicka's[14], 66% were below 610 mg.; and in Fontecella and Sepulveda's[3] series of 20 cases, 85% were below 580 mg. However, Stewart[13] found the chlorides to be normal in most cases. *The Human Cerebrospinal Fluid*[10], in a series of 31, found an average of 625 mg., which is lower by 100 mg. than normal cerebrospinal fluid. The first chloride determination, in the case reported above, was done 7 days after admission; it was found to be 605 mg. per 100 c.c. There was a slight increase on the next two readings; they were 611 and 646 mg. per 100 c.c., respectively.

CONCLUSIONS

1. Tuberculous meningitis has an insidious onset and, even with the clinical picture already established, the cerebrospinal fluid changes may be slight.

2. The cerebrospinal fluid may change rapidly in a few days from one which is almost normal to one characteristic of the disease.

3. The temperature may be normal through part, or throughout the entire course of the disease, as reported by other authors.

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EARLY AMERICAN EXPERIENCE UNDER THE CORONER LAWS

BY TIMOTHY LEARY, M.D.*

In the preparation of a report on the Massachusetts Medical Examiner System for the Rockefeller Foundation's international symposium on Institutes of Legal Medicine† the following historic data were unearthed. As the records deal largely with New England, and many items were discovered which had not appeared in medical collections previously published, their presentation in the columns of the NEW ENGLAND JOURNAL OF MEDICINE seemed appropriate.

THE COLONY OF NEW PLYMOUTH

PREVIOUS to the year 1627, when Governor Bradford put pen to the records, nothing seems to have been recorded; for the early entries in the first volume of deeds relating to garden plots laid out in 1620, the division of land for continuance made in the spring of 1623-1624 and the division of cattle in 1627 were unquestionably written by him in the last-mentioned year. Court records which have been preserved embrace the period from 1633 to 1691 inclusive, with the exception of a few years during the time of the usurpation of Andros[1].

The first record of an inquest pictures the hardships which the early settlers had to undergo, and is as follows:

"At a General Court held the first of March in the Eleventh Yeare of the Raigne of our Soveraigne Lord, Charles, etc. (1635).

At the same Court a jury of twelve being impaniled and charged, in the month of February foregoing to enquire after the death of John Deacon, in the behalfe of our soveraigne lord, the King, gave in their verdict, as followeth, in their owne words, under their own hands, vist:

Having searched the dead body, we finde not any blowes or wounds, or any other bodily hurt. We finde that bodily weakeness caused by long fasting and weariness, by going to and fro, with the extreme cold of the season, were the causes of his death."

The first account of the election of a coroner appears in the record of the general court held the third day of January, 1636, when the annual election occurred. Governor Bradford was reëlected and William Collier, who was elected as one of the governor's "assistants," was also chosen coroner.

THE COLONY OF MASSACHUSETTS BAY

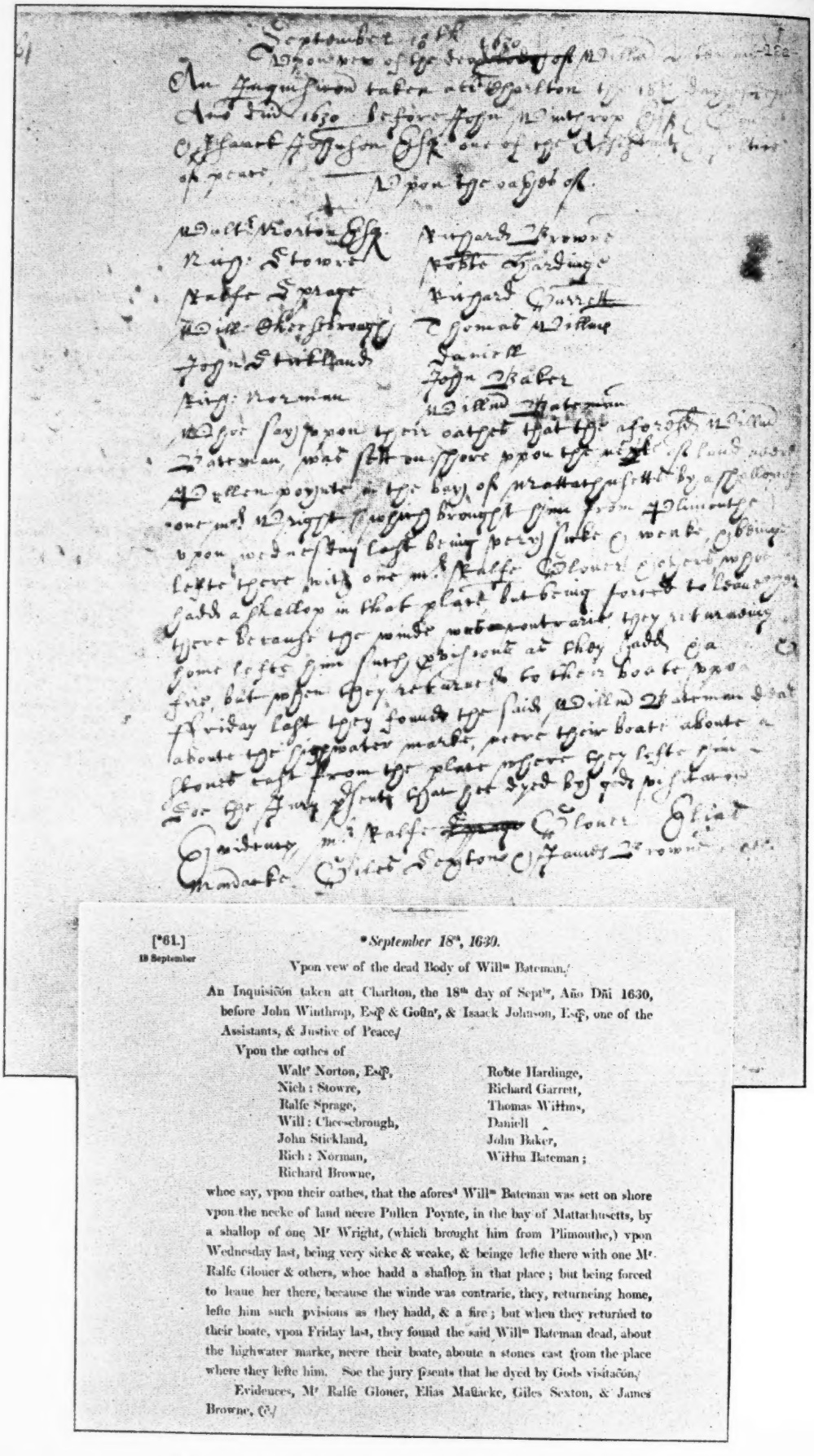
In Figures 1 and 2 are shown photostatic copies of the first inquests recorded in the new colony. The records of the governor and company of the Massachusetts Bay colony began, after the actual landing in Boston, on August 23, 1630. The first of these inquests was held on September 18, 1630, or less than a month following the first colonial records made.

A search of the records of the Colony of Massachusetts Bay discloses no memoranda dealing with the appointment or election of coroners. In 1658, however, there appear twenty-six sections dealing with the duties of constables[2], Section 22 reading as follows:

"In case of untimely or unnatural death of any person to summon a jury of twelve discreete men,

†Methods and Problems of Medical Education, Ninth Series, 1928, p. 297-328.

*For record and address of author see "This Week's Issue," page 786.



[*61.] 19 September

September 18th, 1630.

Vpon vew of the dead Body of Willm Bateman.

An Inquisicõn taken att Charlton, the 18th day of Septbr, Año Dñi 1630, before John Winthrop, Esq & Goũr, & Isaack Johnson, Esq, one of the Assistants, & Justice of Peace.

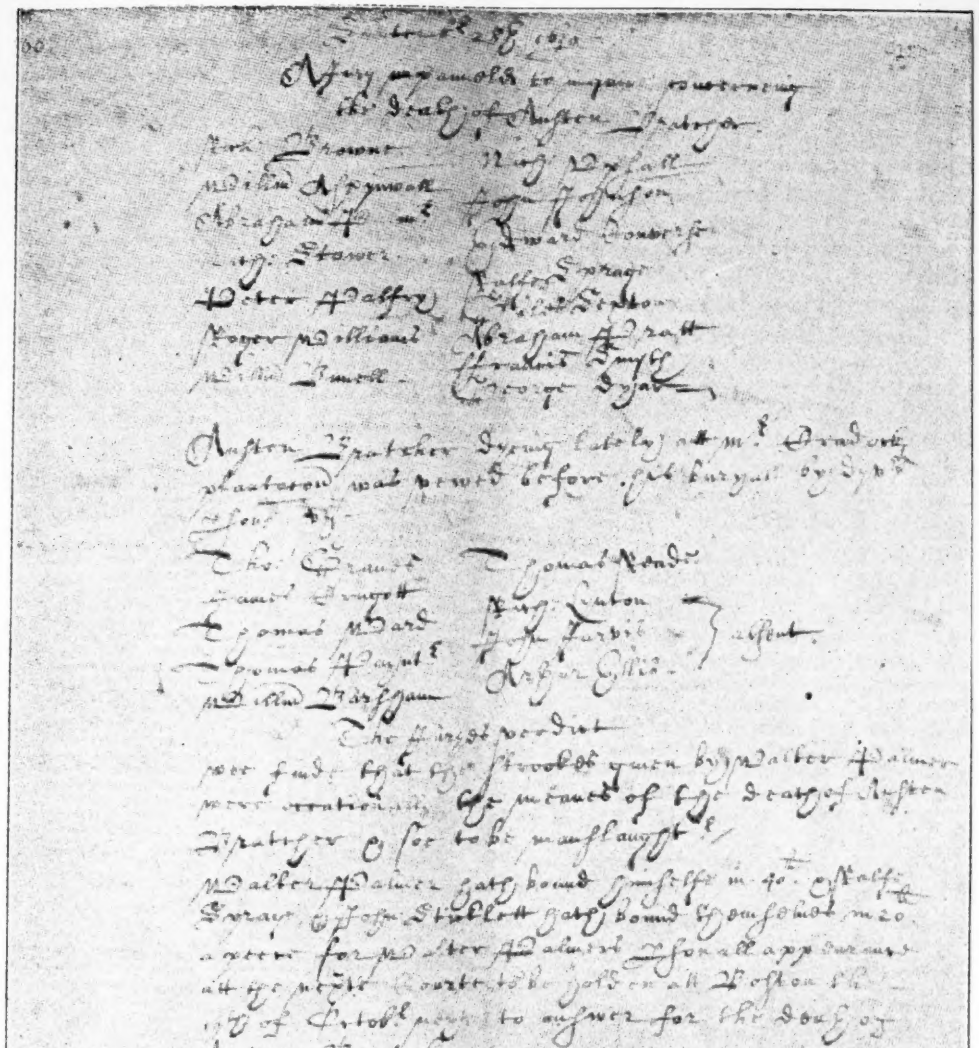
Vpon the oathes of

Walt[r] Norton, Esq,	Robte Hardinge,
Nich: Stowre,	Richard Garrett,
Ralfe Sprage,	Thomas Willms,
Will: Cheesebrough,	Daniell
John Stickland,	John Baker,
Rich: Norman,	Willm Bateman;
Richard Browne,	

whoe say, vpon their oathes, that the afores[d] Willm Bateman was sett on shore vpon the necke of land neere Pullen Poynte, in the bay of Mattachusetts, by a shallop of one Mr Wright, (which brought him from Plimouthe,) vpon Wednesday last, being very sicke & weake, & beinge lefte there with one Mr Ralfe Glouer & others, whoe hadd a shallop in that place; but being forced to leaue her there, because the winde was contrarie, they, returneing home, lefte him such pvisions as they hadd, & a fire; but when they returned to their boate, vpon Friday last, they found the said Willm Bateman dead, about the highwater marke, neere their boate, aboute a stones cast from the place where they lefte him. Soe the jury ꝑsents that he dyed by Gods visitacõn.

Evidences, Mr Ralfe Glouer, Elias Maßacke, Giles Sexton, & James Browne, &c.

FIG. 1. The First Inquest in the Colony of Massachusetts Bay.



* *Septembr 28th, 1630.* [*60.]
28 September.

A Jury impannelld to inquire concerning the Death of Austen Bratcher:—

Rich: Browne,	John Johnson,
Willm Aspinwall,	Edward Converse,
Abraham Palm[r],	Ralfe Sprage,
Nich: Stower,	Giles Sexton,
Peter Palfrey,	Abraham Pratt,
Roger Williams,	Francis Smyth,
Willm Bunch,	George Dyar,
Nich: Upsall,	

Austen Bratcher, dyeing lately att Mr Cradocks plantacon, was vewed before his buryall by dyvr psons, viz:—

1630. 28 September.

Tho: Graves,	Thomas Reade,	
James Crugott,	Rich: Lyston,	
Thomas Ward,	John Jarvis,	Absent.
Thomas Paynt,	Arthur Ellis,	
Willm Barsham,		

The Jurves Verdict:—

Wee finde that the strookes given by Walter Palmer were occationally the meanes of the death of Austen Bratcher, & soe to be manslaught[r].

Walter Palmer hath bound himselfe in 40[li], & Ralfe Sprage & John Stickfast hath bound themselves in 20[li] a peece, for Walter Palmers psonall appearance att the nexte Courte, to be holden att Boston the 19th of Octob[r] nexte, to answer for the death of Austen Bratcher.

FIG. 2. The Second Inquest in the Colony of Massachusetts Bay.

to enquire the cause and manner of theire death, who shall give in a true verdict thereof to the next Assistant or Court of yt shire, on oath."[3]

One is led by this finding and the absence of records of coroners to assume that the coroner's duties were carried out by the constables.

In 1692, when the province of Massachusetts Bay took over the Massachusetts Bay and New Plymouth colonies, the first acts of the legislature were to adopt and legalize temporarily the local colonial laws. Gradually through the following years a new body of laws was established and codified. The first record of coroner laws appears in the Acts and Resolves of the Province of Massachusetts Bay for 1700-01[4]. Chapter three is made up of "An Act relating to the Office and Duty of the Coroner." Under this act the coroner substitutes for the sheriff, when the sheriff is a party to any process. His other duties are limited wholly to the investigation of deaths by violence. There is detailed the form of oath which the coroner is required to take, whose quaint verbiage justifies its reproduction:

"You swear that well and truly you shall serve our soveraign lord the King in the office of a coroner, and as one of his majesty's coroners of the county of S., and therein you shall diligently do and accomplish all and everything and things appertaining to your office after the best of your cunning, wit, and power, for the profit and good of the inhabitants within the said county, taking such fees as you ought to take by law, and not otherwise. So help you God."

EARLY RECORDS OF POST-MORTEM EXAMINATIONS

The first record of post-mortem examinations in America is probably that contained in Champlain's Voyages[5].

"Il y eut de la maladie de la terre parmy nos gens, mais non si aspre qu'elle avoit esté aux annees precedetes: neantmoins il ne laissa d'en mourir sept; & un autre d'un coup de flesche qu'il avait receu des sauvages au port Fortuné.

Notre chirurgien appelé maistre Estienne, fit ouverture de quelques corps, & trouva presque toutes les parties de dedans offensees, comme on avoit fait aux autres les annees precedentes. Il y en eut 8 ou 10 de malades qui guerirent au printemps."

Early records of English colonial post-mortem examinations are few and hard to find. John Eliot, apostle to the Indians, in a letter to the Rev. Thomas Shepard, dated September 26, 1647, bewails the ignorance of the Indians of "Physick," discusses their dependence upon their Powwaws (medicine men), whose methods disgust him, and suggests that if the natives were better instructed and treated medically, they would more readily turn to God. He recommends the establishment of an institution where instruction might be given them. This is the first American allusion to the value of medical missionaries. The final paragraph of the medical portion of this letter reads:

"There is also another reason which moves my thoughts and desires this way, namely that our young students of Physick may be trained up better than yet they bee who have only theoreticall knowledge, and who are forced to practice before ever they saw an Anatomy made or duely trained up in making experiments, for we have never had but one Anatomy in the Countrey which Mr. Giles Forman (now in England) did make and read upon very well." . . .

Whether due to Eliot's influence, or not, the General Court of Massachusetts Bay made the following recommendation, October 27, 1647: "We conceive it very necessary yt such as studies phisick or chirurgery may have liberty to reade anatomy & to anatomize once in foure yeares some malefactor in case there be such as the Courte shall alow of."

An interesting case with an autopsy (incomplete) appears in the Records of the Court of Assistants of the Massachusetts Bay[6],

"4:10 mo, 1654.

We whose names are under ritten; being Called to veue the Body of Matthew Kehnige and to make in Quiery of the suddiness of his Deth and the cause ther of

by searching of his body we finde on his heade on the left side, a wounde which wounde we sawe oppened and ther was corupt blude: and towe small holes out of which Blude Eissued forth and by what we sawe and by the witnesses Brought in on oth we finde that wounde on his heade as neare as we can Judge was a cause of his death.

James Everill	Peter Pl (ace) his (P) mark
Samuell Bidfilld	
	Daniel turell
Nathaniel Wales	
	Samuel (S) Sendall
Barnabas ffawer	his mark
John Phillips	Godfrey (A) Armitage his mark
James Matlocke	
hugh Drury	Henry Blague."

A second deposition more complete but injured by time reads:

"4-10-1654.

Whereas we whose names are hereunder written were required to take a view of ye Body, & especially of ye head of Matthew Cannedge (sodainely) deceased. This is to certefye in dilating the left side of ye head, much corrupt & contused bloud came fort. So the skul being made naked or bare there seemed to be a little cracke by, or through wch cracke, out of it issued a little thin [blood although] away fro ye skul yet notwithstanding it [kept] wheezing forth. And wheras ye sd Mathew [ ] ednes, or blacknes in ye face, the said blacknes much diminished after ye corrupt bloud was come forth by reason of ye said incission of ye head. So yt we conclude it is possible that ye said Mathew his dayes might be shortned by reason of that stroke wch is reported was given him uppon ye head wth ye hamer

Comfort Starr

I doe agree to what is above riten, and also furder doe think it not only possible but very likely to be the cause of his death

John Clark

mr: Clark and mr Starr both were sworne to the truth of ye premis [es] before me

7-10-1654 Ri. Bellingham Govr"

It appeared from the depositions of witnesses that Kehnige or Cannedge, who was an old man, was the master of a fishing boat at Monhegan,

and that Gregory Cassell and John Short were the crew. The depositions indicate that Cassell struck Cannedge on the head with a hammer while both were ashore at Monhegan. Though bleeding freely, Cannedge helped load his vessel with fish and sailed to Boston, where, after an illness, he died. The grand jury indicted Cassell

"for not having the feare of God before his eyes and being seduced by ye instigation of the divill did in or about the middle of October 1[654 by] wound the head of Mathew Cannidge & divers [times] beate ye said Cannidge so as that the sd Cannidge dyed. [ ] wound as Appears by severall evidences, verdict of a (jury) of Inquest given in 4th 10 — 1654. Contrary to the law [es of God] and this Jurisdiccon mo:" etc.

The case came before the Court of Assistants in September, 1657 and was probably transferred to the General Court at its session of October, 1657. At that time the makers of the chief depositions against Cassell, who were fishermen, were out of the jurisdiction, and Cassell was discharged on his own security of one hundred pounds to appear on notice.

In the Colonial Records of Connecticut[7], 1663, March 11, Mr. Rossiter was allowed twenty pounds "in reference to opening Kellie's child and his paynes to visit the Dep. Governor, and his paynes in visiting and administering to Mr. Talcot." Whether the "opening" was ante- or post-mortem, deponent sayeth not.

In the records of the first church in Roxbury[8] it is chronicled that: 1667, March 27. "Mr. John Alcock, Physician, dyed at Roxbury, Mass. His liver was dryed up and became schirrous."

In the same records for 1674 is the following:

"Moneth 6, Day 20—John Bridge died of Ye Wynde Collic and was buried the day following. His body was opened. He had sundry small holes in his stomak and bowels and one hole in his stomak yt a man's fist might passe through, wch is thought was rent wth vyolent straining to vomit the night before he dyed, for the watchers observed yt something seemed t rent wthin him, and he said of it I am a dead man."

The interpretation of these findings is as difficult as is the determination of the cause of death from autopsy protocols made by untrained observers today.

John Josselyn in *An Account of Two Voyages in New England*[9], reports the following case:

"A young maid that was troubled by a sore pricking at her heart, still as she leaned her body or stept down with her foot to one side or another; this maid during her distemper voided worms of the length of a finger, all hairy with black heads; it so fell out that the maid dyed; her friends desirous to discover the cause of the distemper of her heart, had her opened, and found two crooked bones growing upon the top of her heart which as she bowed her body to the right or left side would jab their points into one and the same place till they had worn a hole quite through."

THE GILES COREY CASE

An example of the far reaching harmful consequences of the often hasty and ill-digested findings of a coroner's jury is presented in the case of the famous Giles Corey[10]. Corey came to Salem Town in the early days of its history and became an owner of property. He moved to Salem Village in 1659 and built a house. He was of a somewhat quarrelsome nature and did not get along with his neighbors. His name appears frequently in the Essex County Court Records as a defendant or complainant, particularly in connection with controversies with John Proctor, a neighboring tavern keeper. In 1676 one Jacob Goodale, half-witted son of one of Corey's neighbors, and who was a servant on the Corey farm, had been thrashed by Corey. He was brought home by Mrs. Corey and died several days later. An inquest upon the death was held. The jury filed their findings with Judge William Hathorne[11] as follows:

"We being chosen by the Constable to view the body of Jacob Goodale to see if we can apprehend the cause of his death, we finde several wrongs he hath had in his body as upon his left arme and upon his right thigh a great bruise wch is very swold, and upon the reines of his backe in colour differinge from the other parts of his body we caused an incision to be made much bruised and Run with a gelly and the skin broke upon the outside of each buttocke."

The findings include no record of a verdict.

The incision was evidently made by Dr. Zerobabel Endicot, who appears as a witness before the Quarterly Court later. He was not a member of the coroner's jury, whose names are listed in the record of the findings.

The records of the trial before the quarterly court[12] indicate that Corey had beaten Goodale with a stick. There was evidence that he had also been struck at another time by John Parker, and that he was beaten by a brother, Zacchary Goodale, who caught him stealing apples in his cellar. The court on all the evidence found Corey guilty of assault, and fined him. There the matter rested until 1692—sixteen years later, during the witchcraft period.

Explanations of the witchcraft "disease" which afflicted Salem Village are difficult to arrive at, but a condensed account of the affair is essential to an understanding of the tragic end of Corey, and the use to which the findings of the coroner's jury was put at that time.

Beginning with Roger Bacon, who was accussed of witchcraft because of his learning in the thirteenth century, trials and executions for witchcraft were frequently held in England and Europe during the succeeding centuries. The largest English series was the result of the activities of Matthew Hopkins, "Witch-finder General", who caused the deaths of more than sixty persons in one year, in one county, in England, in 1645-1646, before his exposure. Three "witches" had been executed in Boston: Margaret Jones, June 15, 1648, under Governor

John Winthrop's orders; Ann Hibbins, June 19, 1656, under Governor John Endicott's orders; and a woman named Glover in 1688.

The Glover woman was executed for bewitching four children, all under thirteen years of age. One of the children was a remarkable actress, and succeeded in so impressing Reverend Cotton Mather, that he took her into his house, made an exhaustive study of her actions, and published a sermon on the subject[13], which was widely read. This sermon was reprinted in London, with a preface by Richard Baxter, who declared that he who will not be convinced by all the evidence Dr. Mather presents that the child was bewitched "must be a very obstinate Sadducee."

This was the situation when a group of children who were accustomed to gather at the house of Samuel Parris, minister of Salem Village, began to undergo writhings and groanings, claimed that they were being pinched, beaten, and choked by various persons who were not present in the body.

Upham, who was the historian of "Salem Witchcraft"[14] believes that the detailed story of the actions of the child described by Cotton Mather led the children of the Parris group to attempt to re-ënact her performance. Others accuse Tituba, an Indian woman servant in the Parris family, of being responsible for the children's actions. Tituba, who was later imprisoned, claimed that she accused persons of witchcraft only because she was beaten and abused by her master, unless she made the accusations.

However, as the result of the children's accusations several persons were arrested. The matter drew the attention of Cotton Mather and the General Court at Boston. A special trial court was impaneled and so-called trials were held, as the result of which hundreds of persons were indicted, nineteen were executed by hanging, and Giles Corey was "pressed to death." Corey's death is believed to have had an important influence upon the termination of the persecution.

The witnesses in all of the cases were the group of children and their friends, led by Ann Putnam. Her deposition in the Corey case is so characteristic of the testimony against all of the accused that I quote it:

> "The deposition of Ann Putnam who testifieth and saith that cn the 13th of April 1692 I saw the Apperishtion of Giles Corey com and affiect me urging meto writ in his book and so he continued hurting me by times tell the 19th April, being the day of his examination and during the time of his examination Giles Corey did tortor me a grat many times: and also severall times sence Giles Cory or his apperance has most greviously afflected me by beating, pinching, and almost choaking me to death urging me to writ in his book also on the day of his examination I saw Giles Cory or his apperance most greviously affiect and torment mary walcott mercy lwews and sarah vibber and I verly believe Giles Cory is a dreadfull wissard for sence he has ben in prison he or his Apperance has come to me a grat many times and afflected me." (Owned under oath, Sept. 9-92.)

Mary Walcott, Mercy Lewis, and Sarah Vibber were part of the "group," who testified in most of the cases.

The activities of the courts, which had been progressive up to Corey's case were disturbed by the fact that Corey refused to plead. Many of the accused had plead guilty, and, while they were imprisoned and their property confiscated, none of them were executed.

Only the strong characters, armed in righteousness, dared to plead not guilty, and had been in large part executed, and their property forfeited. Corey, eighty-one years of age, refused to plead, since his plea, whether of guilty or not guilty, would have been followed by the confiscation of his estate. As a result of his standing mute he was ordered to undergo the ordeal of *peine forte et dur*. "He was put into a low and dark apartment in the prison. He would there be laid on his back on the bare floor, naked for the most part. A weight of iron would be placed upon him, not quite enough to crush him. He would have no sustenance, save only, on the first day, three morsels of the worst bread; on the second day three draughts of standing water that should be nearest to the prison door; and, in this situation, such would be alternately his standing diet till he died, or till he answered."[15]

This order of the court was carried out. The sturdy old hero lived for several days under the ordeal and made a will, leaving his property to the sons-in-law who had stood by him during his persecution. The death of the old man under the torture profoundly affected the public mind, and it was felt by the supporters of the persecution that some manifestation was necessary to renew the general infatuation. Ann Putnam, the dependable witness, came forth with the required revelation. On the day of Corey's death Judge Sewall, then in Salem, received from Thomas Putnam, her father, a letter to this effect:

> Last night my daughter Ann was grievously tormented by witches, threatening that she should be pressed to death before Giles Corey: . . . there appeared to her (she said) a man in a winding sheet, who told her that Giles Corey had murdered him by pressing him to death with his feet; but that the Devil there appeared unto him, and promised that he should not be hanged. The apparition said God hardened his heart, that he should not hearken to the advice of the court, and so die an easy death; because, as it said, it must be done to him as he has done to me. The apparition also said that Giles Corey was carried to the Court for this, and that the jury had found the murder; and that her father knew the man and the thing was done before she was born[16].

Cotton Mather in his book *The Wonders of the Invisible World, being an Account of the Tryals of Several Witches lately Executed in New England, and of several Remarkable Curiosities therein occurring*[17], comments upon the Corey case, under the heading, "A Fourth Curiosity." He accepts the Ann Putnam story as a true

revelation, since the death of Goodale occurred before Ann's birth, and the child could not have heard of the case. When it is remembered that Salem Village was an isolated village with only 123 houses and families in 1692, the ingenuousness which would accept this evidence, that the child had never heard of what must have been a *cause celèbre* in village history, is monumental. Mather continues:

For all People now remember very well (and the Records of the Court also mention it) That about Seventeen Years ago *Giles Corey* kept a man in his House, that was almost a Natural Fool; which Man died Suddenly: A Jury was Impannel'd upon him, among whom was Dr. Zerobabbel Endicot; who found the Man bruiz'd to Death, and having clodders of Blood about his Heart. The Jury, whereof several are yet alive, brought in the Man Murdered; but as if some Enchantment had hindered the Prosecution of the Matter, the Court proceded not against *Giles Corey*, tho it cost him a great deal of Mony to get off.

No comment is necessary upon the easy transition from beaten to bruised and from bruised to pressed, or the evident misquotation of facts.

The horrible death of Corey and the arrogance of the persecutors, who caused the children to accuse of witchcraft all who criticized the persecution, even to ministers, their wives, and the wives of officials, brought to a termination this delusion, and the terror which marked its progress.

A second inquest in the Essex jurisdiction is reported May 2, 1678[18].

"Sr According to your order I with the rest of the men whose names are under writne have searcht the Body of one called Edward Bodge. I made Incision upon the parte of his Body which was most suspitious which was upon the Temporall musile: I layd the Bone Bare: wee could nott find any fracture in the least nether was the flesh in anywise corupt or putrified. Marblehead, Octobr 31th 1678. Richard Knott Chir., and Christopher Lattimor, Ambros Gall, Robert Bartlett and John Merrett."

The diary of Judge Samuel Sewall[19], one of the judges in the Salem witchcraft trials, who later recanted and publicly confessed his guilt, and who has been called the Pepys of the American Colonies, records: "Sept. 22, 1676. Spent the day from 9 in the M. with (Dr.) Brakenbury, Mr. Thompson, Hooper, Cragg, Pemberton, dissecting the middlemost of the Indians executed the day before. X (Hooper) who, taking the ♡ in his hand affirmed it to be the stomak. I spent 18s 6d in Madiera wine and 6d I gave to the maid."

In the Records of the First Church in Roxbury, Mass.[20], by Rev. John Elliot, we find the following entry:

1674-25-10m

"being desired I went to Watertowne to be present at the cuting b. livermore's daughter of a wonderful great timpany. the operation succeeded at the present blessed be the lord. 28. 10m the above named woman cut of a timpany, the next day dyed, there were about 15 gallons of water taken from her p'tly afore she dyed, the rest after she was dead."

In the diary of John Dennison Hartshorn, in the possession of the Boston Medical Library, there is a record of the duties and experiences of this sixteen year old boy, who served his apprenticeship with Dr. Silvester Gardiner in Boston, from August 1752 to his death in May 1756. The following extract is interesting from our standpoint.

"Nov. 19 (1752) was called to Fort Hill to dress Darby boy and who was stabbed with a long knife by Wm. Welsh. The intestines were two feet out of the wound, cut almost asunder in places. After performing the Glover's suture, reduced 'em first enlarging the external orifice. The man died in 24 hours, after receiving the wound. Was carried to the almshouse and there dissected."

THE CASE OF GOVERNOR SLOUGHTER

In 1688-1689, when Governor Andros was disposed of by the Bostonians, there was left in charge of the Province of New York Lieutenant-Governor Nicholson, who became an object of suspicion to the people from his connection with the unpopular government of James II. When tidings of the access of William of Orange to the throne reached the colony the common people, distrustful of Nicholson and his powerful official following, seized the fort with the public moneys, and turned the key over to Jacob Leisler to preserve for the representative of King William. Leisler promptly proclaimed William, by sound of trumpets, King of England and the Colonies. Nicholson and his council demanded the moneys and appointed three commissioners to receive the revenues. The people revolted, and overcame the commissioners, Nicholson fleeing to England and Bayard to Albany. From that time Leisler acted as lieutenant-governor of New York. Henry Sloughter was appointed governor of New York province by King William in 1689. He did not appear in the province until 1691. Captain Richard Ingoldsby came to New York in advance of the new governor. Ingoldsby refused to show his credentials and Leisler refused to surrender his authority to him. On the arrival of Governor Sloughter, March 19, 1691, Leisler delivered over the government to him. Leisler and nine associates were arrested, tried as rebels by a special commission of Oyer and Terminer, made up of Leisler's enemies and Sloughter's followers, and convicted of treason. Sloughter, who was addicted to alcohol, is said to have signed Leisler's death warrant while drunk. Following Leisler's death, Sloughter died suddenly after a drinking bout, and accusations were made that he was poisoned by a negro employed by Leisler's friends, who put poison in his soup. The council ordered a post-mortem examination, which was made by Dr. Johannes Kerfbyle and five other physicians.

Toner, who saw the original report of the physicians in the New York State papers, states that the examination was a most complete one. The fire in the capitol at Albany in 1911 destroyed many manuscripts, and among them apparently

this report, which is not in the state manuscripts now.

The calendar of Council Minutes[21] carries the following record:

The Doctors and Chirurgeons appointed to view the late Governor's body pursuant to the order of this board returned under their hands and seals, That the Governor dyed of a defect in his blood and lungs occasioned by some glutinous tough humour in the blood which stopped the passages thereof and occasioned its settling in his lungs which by other accidents increased until it carried him off of a sudden. And for further satisfaction to the Council made Oath that they knew no other Cause of his Death.

Ordered that Eight pounds Eight shillings be payd by Mr. Collector for opening and inspecting the said body.

The general agreement is that Sloughter's death was due to alcoholism or pulmonary embolism.

Undoubtedly the most dramatic post-mortem of the colonial period was that performed upon Crispus Attucks, a victim of the Boston Massacre, March 5, 1770, by Dr. Benjamin Church, poet, scholar, orator, statesman, and first surgeon-general of the American Army, who was later convicted of treason. His deposition follows:[22]

"I, Benjamin Church, Jun., of lawful age, testify and say, that being requested by Mr. Robert Pierpont, the coroner, to assist in examining the body of Crispus Attucks, who was supposed to be murdered by the soldiers on Monday evening the 5th instant, I found two wounds in the region of the thorax, the one on the right side, which entered through the second true rib within an inch and a half of the sternum, dividing the rib and separating the cartilaginous extremity from the sternum, and ball passed obliquely downward through the diaphragm and entering through large lobe of the liver and the gall-bladder, still keeping its oblique direction, divided the aorta descendens just above its division into the iliacs, from thence it made its exit on the left side of the spine. This wound I apprehended was the immediate cause of his death. The other ball entered the fourth of the false ribs, about five inches from the linea alba, and descending obliquely passed through the second false rib, at the distance of about eight inches from the linea alba; from the oblique direction of the wounds, I apprehended the gun must have been discharged from some elevation, and further the deponent saith not."

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THE PATHOLOGY OF DIABETES IN YOUNG ADULTS*

BY IRVINE H. PAGE, M.D.† AND SHIELDS WARREN, M.D.†

IN a previous paper[1] the pathology of diabetes mellitus in childhood has been considered. Owing to the small number of cases in which pathological data are available and the importance of studying young and presumably uncomplicated cases of the disease, it seems worth while to place on record the following cases of young diabetics which came to post mortem examination. Those autopsied cases in our series where the onset of the disease occurred between fifteen and twenty-five years of age, and one additional case of juvenile diabetes, are included in the present group. The cases are arranged in order of age at onset of the disease.

CASE I. P H 9949. Female, colored, 12 years of age. Duration of disease four years. Past history essentially negative. Jan. 7, 1925 admitted and placed on diet of C120, P60, F115, with insulin thirty-two units per day, which kept her sugar free. June 8, 1925, admitted in coma. Two weeks previously she had developed a cough. Blood sugar 0.65%. Rapidly recovered under coma therapy. May 26, 1927 admitted in coma. Blood sugar 0.278%, plasma CO_2 17.6 volumes per cent. Recovered rapidly under coma therapy, kept sugar free on diet of C100, P60, F120, with insulin forty-five units per day. Oct. 31, 1927 coma four days after sore throat. Blood sugar 0.43%, plasma CO_2 14.0 volumes per cent. Death thirteen hours after admission. Autopsy findings: an under nourished and under developed negro girl. Focus of confluent bronchopneumonia in right upper lobe with pleuritis. Heart, weight 120 gms. There are numerous small atheromatous plaques along the bases of the aortic cusps, otherwise negative. The aorta shows numerous raised, bright yellow atheromatous plaques about the mouths of the coronary arteries. About the intercostal arteries and in the abdominal portion are numerous small, raised, yellow atheromatous plaques. Liver shows fatty infiltration. Pancreas is negative in gross and microscopically except for early post mortem change. Microscopic examination of the aorta shows extensive lipoid infiltration in the intima with fraying of the internal elastic lamina. The spleen on microscopic examination shows a number of cells containing droplets of fat and the liver shows extensive

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†For record and address of author see "This Week's Issue," page 786.

fatty infiltration and droplets of fat in the Kupfer cells throughout.

CASE II. P H 9388. Female, 16 years old. Duration of diabetes three months. Onset one month after severe cold. Two months after onset convulsive seizures of right hand and right leg lasting three to four minutes. Admitted in deep coma. Blood sugar 0.35%, CO_2 10.5 volumes per cent. Received one hundred and sixty units of insulin, fluid, and glucose, but died. Post mortem examinations essentially negative except for small scattered atheromatous plaques in aorta and a tangled mass of dilated vessels (angioma of pia) over the left cerebral hemisphere. Pancreas normal in gross; microscopically islands abundant. No evidence of hydropic or other degeneration.

CASE III. P H 9292. Male, 17 years of age. Duration of disease two months. Past history essentially negative. Headache for five days. Nausea and vomiting for three days. Coma for twelve hours. CO_2 13.0 volumes per cent., blood sugar 0.76%, urine sugar 3.4%. Died in coma. The significant autopsy findings were rheumatic endocarditis and bronchopneumonia. Pancreas negative in gross and microscopically. Islands appear numerous and show only occasional hydropic cells. One island shows hemorrhage.

CASE IV. P H 8770. Male, 18 years of age. Duration of diabetes fifteen months. Eighteen months before death urine sugar and blood sugar negative. Twice severe acidosis which stopped just short of coma. Shortly before death CO_2 23.7 volumes per cent., blood sugar 0.40%. Death did not occur from coma but apparently from progressive loss of strength and weakness. Autopsy essentially negative except for a small heart (160 gms.) and early bronchopneumonia in the left lung and a greatly dilated stomach containing several liters of partly digested food. The pancreas is normal in gross; microscopically negative except for scarcity of islands. Only four or five islands are seen in examining fourteen sections from different parts of the pancreas.

CASE V. P H 9038. Female, 18 years old. Duration of diabetes eight months. Two weeks before admission symptoms increased in severity in spite of dietary treatment. Blood sugar 0.43%, CO_2 12.6 volumes per cent. Died in coma. Autopsy reveals striking atheromatous plaques of ascending portion of aorta and descending portion of left coronary artery. Numerous bright yellow atheromatous plaques scattered along the aorta, particularly marked about the opening of the intercostal arteries. Liver negative in gross; microscopic examination shows fatty infiltration and absence of glycogen except for a few infiltrated nuclei. Pancreas normal in gross and microscopically.

CASE VI. 2720. Male, 19 years of age. Duration of diabetes 0.9 years. Blood sugar 0.30%. Alveolar CO_2 19 volumes per cent. Cause of death perinephric abscess. Post mortem examination essentially negative except for the presence of large perinephric abscess and glycogenic infiltration of kidney tubules and of nuclei of liver cells. Pancreas is small but normal in gross. Microscopically the only change is a moderate degree of lymphatic infiltration of the islands.

CASE VII. P H 9347. Female, 20 years old. Duration of diabetes six months. Five days before death caught cold and brought to hospital in coma. Blood sugar 2.38%, CO_2 9.2 volumes per cent. Given large amount of insulin, glucose, and caffein, but died thirty hours after admission. Post mortem examination shows marked tortuosity of coronary vessels with numerous yellowish atheromatous plaques. The ascending aorta also shows numerous orange-yellow atheromatous plaques. Right lung shows confluent bronchopneumonia. Pancreas negative in gross. Islands normal in number. Some islands show a number of compactly arranged cells tending toward the columnar form, somewhat smaller than normal.

CASE VIII. P H 8759. Male, 25 years of age. Duration of diabetes three years. Blood sugar 0.179%, CO_2 46.2 volumes per cent. Day before death sank into coma although alveolar CO_2 remained high (40 volumes per cent.), temperature 94° F. Post mortem examination shows the heart weighing 210 gms. with numerous atheromatous plaques about the mouth of the coronary arteries. The aorta shows numerous atheromatous plaques which are evenly distributed down to the bifurcation. The iliac and femoral arteries show marked thickening of their walls. No calcification. Diffuse bronchopneumonia involving both lungs. Pancreas negative in gross. Islands markedly reduced in number. On six sections only four islands can be seen although there are many small stellate scars suggesting obliteration by fibrosis of previously existing islands. Microscopic examination of the femoral arteries shows marked thickening of the intima due to cellular connective tissue with some fatty infiltration.

CASE IX. P H 8971. Female, 23 years old. Duration of diabetes sixteen months. Past history essentially negative. Lost thirty pounds in last sixteen months. Blood sugar 0.243%. On diet C60, P75, F90 became sugar free and was discharged. One year later admitted for lobar pneumonia CO_2 27.1 volumes per cent. Post mortem examination essentially negative except for numerous fibrous patches in the myocardium, tortuous cord-like coronary arteries and scattered atheromatous plaques along the course of the pulmonary artery and the aorta. The left lower lobe of the lung is completely consolidated. Pancreas negative in gross; microscopically it shows islands normal in number and in condition. Slight interstitial fibrosis in the acinar tissue.

CASE X. 1907. Male, 24 years of age. Duration 1.8 years. Blood sugar 0.44%, alveolar CO_2 31 volumes per cent. Died in coma. Post mortem examination is normal except for a small pancreas showing moderate hyaline degeneration of the islands of Langerhans and moderate sclerosis of the acinar tissue. There is glycogenic infiltration of the kidney tubules.

CASE XI. 1794. Female, 33 years of age. Duration of diabetes eight years. Patient has been under insulin treatment for three years, the weight increasing from sixty-seven to one hundred thirty-five pounds. She had had five Stokes-Adams attacks. She died in attack of sudden pain in chest and left arm. Blood sugar 0.22%, alveolar CO_2 28 volumes per cent. Post mortem examination shows fresh infarct of apex of left ventricle and interventricular septum. There are atheromatous plaques on the endocaridum below the aortic valve. Coronary arteries show numerous atheromatous plaques and the left barely admits a horsehair. Aorta shows extensive atheromatous changes. Pancreas is negative in gross; microscopic examination shows the islands to be decreased in number and with various degrees of fibrosis. The spleen shows marked infiltration with fat, most of which is contained in large mononuclear leukocytes scattered throughout the pulp and the germinal centers.

(Blood sugar and CO_2 determinations taken from one to forty-eight hours before death.)

DISCUSSION

The most striking feature in this group of cases, as in juvenile diabetics, is the slight amount of pathological change that can be detected in the pancreas. One of the cases, Case IV, showed marked reduction in the number of islands. Three of the older individuals in this series exhibited fairly marked pathological change in the islands. In Case VIII there was extensive sclerosis of the islands. Case X showed moderate hyaline degeneration of the islands, and Case XI a moderate amount of fibrosis of the islands together with some decrease in number.

The outstanding pathological change in this group is the evidence of disturbance of lipoid metabolism, particularly as manifested in atheromatous changes in the blood vessels. In previous papers this point has been stressed and the present group of cases affords still further evidence of the striking amount of atheromatous changes present even in young diabetics. In Case I, for example, only 12 years of age, the atheromatosis and the changes in the internal elastic lamina were comparable to those of a 50 or 60 year old man. This case also, as did Case XI, showed other evidence of disturbed lipoid metabolism—the presence of lipoid droplets in the reticulo-endothelial cells of the spleen and liver. Case XI, 33 years of age, died of an infarct of the heart secondary to sclerosis and almost complete occlusion of the coronary arteries together with extensive atheromatous degeneration of the aorta. Case VIII also showed striking atheromatous degeneration of the aorta with marked intimal thickening of the iliac and femoral vessels. Less marked atheromatosis was shown by cases II, V, VII, and IX. Thus only four out of eleven cases showed no evidence of atheromatosis.

These cases of young individuals with extensive arteriosclerosis, or perhaps better, atherosclerosis, emphasize the danger which arterial disease presents to diabetics and forcibly calls our attention to the need for considering the lipoid, as well as the carbohydrate, metabolism of the diabetic.

The pathology of the pancreas in young diabetics is almost invariably a disappointment to the morphologist, and this group is no exception. The younger the individual, the less complicated his diabetes and the less likelihood there is of demonstrable anatomical change in the pancreas.

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SUGAR TOLERANCE AND HYPERTENSION IN CATARACT PATIENTS*

Observations in a Series of One Hundred Cases

BY E. R. BLAISDELL, M.D., F.A.C.P.†

IN many instances, cataract is discovered in patients who also have diabetes or renal vascular disorders, such as essential hypertension, arterio-sclerosis and nephritis. These diseases most frequently occur at a time in life when cataract is most common. With the exception of diabetes, they do not appear to have any direct bearing upon the etiology except that they are not infrequently associated with an increase of sugar in the blood.

Cataract does seem to be directly related to hyperglycemia in some instances. S. Weir Mitchell[1], while injecting large amounts of sugar into dogs, found that cataracts were produced in a number of the animals. Langdon, by reducing blood sugars to normal, completely arrested lens changes for three years in four patients and in one there was improvement in the lenticular condition. Joslin[2] believes that cataracts must have a definite relation to the hyperglycemia, because they occur in young diabetics who have had the disease many years. F. M. Spalding and W. S. Curtis[3] studied 307 successive cases of Joslin's series and found cataracts in 10.4%. Baldwin and Barthell[4], in a series of 132 cases of cataracts, found the fasting blood sugar above .12% in 99 cases.

From Oct. 1, 1926 to Apr. 1, 1928, 100 patients entered the Maine Eye and Ear Infirmary for cataract operations. Through the courtesy of the visiting ophthalmologists, Drs. E. E. Holt, Jr. and S. Judd Beach, these patients were referred to the writer's metabolic clinic for study. A complete history was taken, physical examination made and blood chemistry done on each patient on admission. The average life of all patients was 62.5 years. Patients nos. 4, 21, 48, 56, 92, 93 and 96 had a high fasting blood sugar with heavy glycosuria and were put on pre-operative diabetic treatment. Patients nos. 46 and 80 had received rigid treatment for diabetes before entrance and were referred back to the eye service without further study. The remaining 91 were given 100 grams of glucose on a fasting stomach and blood sugar estimations were taken two hours later.

It was considered that .140% was a high normal blood sugar reading two hours after the test. Patients nos. 8, 14, 71 and 75 showed a fasting blood sugar above .120% and patients

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nos. 71, 76 and 98 showed a blood sugar above .140% two hours after the administration of the sugar. Adding these three cases to the number of known diabetics in the series, there are 12% with faulty carbohydrate metabolism. Thorough physical examinations failed to disclose any cause for this dysfunction other than that of pancreatic deficiency.

Folin-Wu's method for determining blood sugar was used throughout. Benedict's test solution was used for detecting urinary sugar.

Blood pressures were taken on entrance and 150 was adopted as a high normal systolic reading. If the entrance systolic blood pressure was above this mark, it was not recorded but was taken again the next morning, while the patient was in bed, in order to rule out, as far as possible, the effect of nervous excitement. In 31 patients there was a systolic blood pressure above 150. This figure is not high when one takes into consideration that the average age of all patients was over 60 years and that Fahr[5]

Case No.	Blood Pressure	Urine before 100 gms. of Glucose	Urine after 100 gms. of Glucose	B. Sugar before 100 gms. of Glucose	B. Sugar after 100 gms. of Glucose
1.	125/80	0	0	.100%	.130%
2.	130/80	0	0	.086%	.100%
3.	168/98	0	0	.118%	.121%
4.	200/110	2%	—	.220%	—
5.	120/70	0	0	.080%	.107%
6.	110/80	0	0	.080%	.111%
7.	140/80	0	0	.102%	.142%
8.	150/90	0	0	.133%	.140%
9.	150/90	0	0	.100%	.114%
10.	160/80	0	0	.101%	.121%
11.	150/70	0	0	.120%	.133%
12.	130/70	0	0	.111%	.122%
13.	150/80	0	0	.118%	.127%
14.	160/90	0	.2%	.108%	.182%
15.	200/110	0	0	.111%	.133%
16.	140/78	0	0	.093%	.121%
17.	100/70	0	0	.076%	.101%
18.	140/98	0	0	.111%	.140%
19.	140/80	0	0	.107%	.121%
20.	145/80	0	0	.118%	.130%
21.	140/90	4%	—	.210%	—
22.	140/40	0	0	.093%	.118%
23.	140/60	0	0	.108%	.123%
24.	150/60	0	0	.111%	.123%
25.	158/60	0	0	.111%	.128%
26.	200/110	0	0	.107%	.127%
27.	200/100	0	0	.076%	.085%
28.	150/78	0	0	.076%	.090%
29.	198/90	0	0	.100%	.118%
30.	150/80	0	0	.098%	.113%
31.	140/80	0	0	.110%	.121%
32.	140/80	0	0	.096%	.118%
33.	150/80	0	0	.083%	.120%
34.	140/80	0	0	.109%	.120%
35.	120/75	0	0	.095%	.114%
36.	160/40	0	0	.118%	.121%
37.	90/60	0	0	.083%	.099%
38.	120/60	0	0	.098%	.111%
39.	140/80	0	0	.098%	.114%
40.	138/95	0	0	.097%	.120%
41.	138/74	0	0	.097%	.118%
42.	120/70	0	0	.103%	.118%
43.	140/80	0	0	.098%	.111%
44.	200/95	0	0	.118%	.120%
45.	190/100	0	0	.092%	.111%
46.	150/80	0	—	.110%	(Known diabetic)
47.	140/80	0	0	.106%	.121%
48.	230/140	5%	—	.197%	
49.	130/80	0	0	.111%	.122%
50.	150/80	0	0	.109%	.124%
51.	130/80	0	0	.104%	.128%
52.	190/111	0	0	.114%	.130%
53.	140/80	0	0	.098%	.111%
54.	130/70	0	0	.100%	.127%
55.	160/90	0	0	.107%	.126%
56.	190/110	.4%	—	.220%	—
57.	135/80	0	0	.120%	.133%
58	168/90	0	0	.117%	.137%
59.	150/70	0	0	.117%	.121%
60.	130/80	0	0	.100%	.121%
61.	140/78	0	0	.098%	.120%
62.	220/110	0	0	.095%	.100%
63.	80/50	0	0	.095%	.102%
64.	140/70	0	0	.117%	.125%
65.	140/70	0	0	.107%	.111%
66.	170/80	0	0	.100%	.114%
67.	140/80	0	0	.114%	.125%
68.	140/80	0	0	.114%	.120%
69.	190/100	0	0	.114%	.119%
70.	128/80	0	0	.114%	.122%
71.	160/100	0	0	.137%	.154%
72.	138/78	0	0	.105%	.115%
73.	200/100	0	0	.114%	.119%
74.	220/100	0	0	.113%	.127%
75.	140/78	0	0	.133%	.140%
76.	130/80	0	0	.080%	.150%
77.	148/80	0	0	.095%	.104%
78.	140/80	0	0	.103%	.110%
79.	120/70	0	0	.105%	.125%
80.	140/80	0	(Known diabetic)	.120%	—
81.	130/90	0	0	.064%	.093%
82.	220/100	0	0	.080%	.129%
83.	200/100	0	0	.110%	.133%
84.	160/80	0	0	.100%	.121%
85.	128/70	0	0	.100%	.114%
86.	140/80	0	0	.100%	.127%
87.	150/80	0	0	.090%	.140%
88.	140/80	0	0	.125%	.130%
89.	142/64	0	0	.114%	.125%
90.	169/80	0	0	.083%	.100%
91.	140/80	0	0	.093%	.105%
92.	140/80	2%	—	.245%	—
93.	220/110	3%	—	.278%	—
94.	180/90	0	0	.080%	.100%
95.	137/70	0	0	.114%	.140%
96.	140/80	2%	—	.200%	—
97.	182/100	0	0	.107%	.124%
98.	130/80	0	.3%	.124%	.210%
99.	158/70	0	0	.087%	.099%
100.	130/78	0	0	.093%	.125%

has recently shown that 23% of all deaths in persons 50 years of age and over, is the result of hypertension.

Hypertension and low glucose tolerance do not seem to be contraindications for cataract operations, providing intelligent pre-operative and post-operative treatment is carried out. In this series, there was no patient with delayed healing or post-operative hemorrhage.

The number of cases studied is far too small for one to draw any conclusions as to the etiology of cataract, but the results obtained here, added to the reports from other clinics, indicate that the presence of low glucose tolerance and cataract in the same patient is more than a coincidence. Research workers and pathologists have taught us that certain organs of certain families are susceptible to certain diseases and that this susceptibility does not always show itself in the following generations. Given such a patient, predisposed to cataract, is he not more liable to develop the disease if his carbohydrate metabolism becomes impaired?

SUMMARY

The blood sugar tolerance was studied and the blood pressures recorded in one hundred patients with cataracts. With the exception of nine known diabetics, all were given 100 grams of glucose on a fasting stomach and the blood sugar was estimated two hours later. Twelve patients had a carbohydrate tolerance below normal, while thirty-one patients had a systolic blood pressure above 150 mm.

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WHY A GRADUATE COURSE IN CANCER

For the last three years a very great amount of public attention in Massachusetts has been directed to the problem of cancer. We know that at least 30 per cent. of the cancer cases might be saved if in each instance adequate treatment was applied sufficiently early. The control problem then comes down to bringing the people to the physicans sufficiently early and in having the physicians sufficiently alert to show those needing service where it can be obtained. It is probably true that today in Massachusetts resources for the proper diagnosis and treatment of cancer are more generally available on a wider scale than ever before in the country.

In addition to the hospitals and clinics that have long been developed under private initiative, of late the State has opened a cancer hospital and there have been developed in seventeen cities and towns widespread over the State definitely organized cancer clinics which anyone may attend. This decentralizing of the service has raised a very real problem of maintaining quality. This is being met through constant association of a group of men familiar with cancer in the clinic, the use of consultants, and periodic meetings of the staffs from all these clinics.

The public is responding astonishingly to the instructions regarding early recognition. Following the five day cancer campaign of last year, 12,000 people sought medical advice in a month. 700 cancer patients were seen in the State-aided clinics and we estimate that 9,000 people with cancer were seen by physicians in the State last year. This represents about 90 per cent. of all the persons with cancer in the State that year.

Granted that these figures are only estimates, they are astonishing and sobering. Is the service that these people are getting sufficiently adequate to affect favorably in the years to come our appalling cancer death rate?

The value of the clinics is not so much in the people they actually see as that through their presence in a community a consciousness of the cancer problem is developed in the medical profession. Our campaign last year showed that nearly 95 per cent. of the persons seeking medical advice for cancer sought it in the private office. Should our clinics be completely effective in every case coming to their attention the effect on the cancer death rate would be negligible. Results must be accomplished through service in the private office.

Figures show that if all cancer patients in the State were equally distributed among all the doctors each man would see but two cases of cancer a year. Since cancer is so catholic in its distribution and varies in its manifestations so radically with each organ involved it is of course out of the question to expect in view of these figues that each physician should be thoroughly expert, particularly in the early recognition (which is the most baffling of all), with all types of cancer. The hope, however, is as in early tuberculosis, that the physicians will become sufficiently familiar with the early signs which should indicate those persons who must be sent where resources for complete diagnosis and adequate therapy are available.

With this in view, the Massachusetts Medical Society is offering to the physicians of the State a graduate course in cancer from April 23rd to 25th. Here in a short space of time will be offered in a more complete form than ever before an outline of what in view of our present knowledge is adequate treatment for each type of the disease and how one shall determine early the cases needing this treatment.

What Massachusetts will accomplish in cancer to a very great extent may be judged by the interest that the profession will show in making use of the opportunity offered in this graduate course.

MEDICAL PROGRESS

PROGRESS IN PEDIATRICS

BY JOHN LOVETT MORSE, M.D.*

ROENTGENOGRAPHIC CHANGES IN THE THORAX OF NORMAL NEW-BORN BABIES

(Weymuller, Bell and Krahulik, *American Journal Dis. Children,* 1928, 35, 837.)

THEY made daily Roentgenographic studies of the thorax of twenty-five normal infants from birth to two weeks. The most striking observation was a well marked definition of the bronchial trees which extended well toward the parenchyma of the lung. There were frequently considerable mottling and peribronchial thickening to an extent which is often interpreted as characteristic of pneumonia. These linear markings were most distinct in the first two days, but persisted throughout the two weeks. In eleven of the twenty-five patients the interlobar pleura between the right upper and middle lobes cast a definite shadow. They found that accurate measurement of the changes in the size of the heart was impossible. There was a fairly regular tendency toward reduction in the size of the cardiac shadow after the second day. The size did not seem to vary after this time. In one apparently normal baby the cardiac shadow was huge on the first day, but gradually diminished, so that after the third day it corresponded with the average. Other studies showed that the width of the upper mediastinal shadow often changes most markedly with respiration and that it may readily be confused with the shadow interpreted as due to an enlarged thymus gland. This is particularly true if but a single picture is taken and that during expiration.

HEMORRHAGE OF THE SUPRARENALS IN THE NEW-BORN INFANT

(Goldzieher and Greenwald, *American Jour. Dis. Children,* 1928, 36, 324.)

Hemorrhage into the suprarenals is more common at or shortly after birth than at any other time. In the majority of cases the diagnosis is impossible, but in some the symptomatology is characteristic. The symptoms vary according to the extent of the bleeding and the damage to the glandular parenchyma. If the hemorrhage is large, the symptoms are primarily those of severe internal bleeding. If the hemorrhage has broken through into the peritoneum, the symptoms of peritoneal irritation are added to those of hemorrhage. If the hematoma is very large, there may be symptoms of pressure on adjacent structures. When the hemorrhage is one of the manifestations of hemorrhagic disease of the new-born, there are usually other evidences of this disease.

In other instances, however, the symptoms are, as in the two cases reported by Goldzieher and Greenwald, those of acute suprarenal insufficiency. At this age, the function of the cortex is far more important than that of the medulla. Consequently, the symptoms are those of cortical insufficiency. The two main activities of the suprarenal cortex are regulation of the temperature and respiration. The two outstanding symptoms, therefore, are hyperpyrexia and dyspnea, the height of the temperature and the degree of dyspnea apparently being in direct proportion to the amount of cortical tissue destroyed. In one of their cases the temperature was 106°F. and the respiration 130. In the other, the temperature was 103°F. and the respiration 60. The presence in a new-born infant of high temperature and rapid respiration without evidences of pulmonary involvement suggests strongly, therefore, hemorrhage into the suprarenals. Associated symptoms are pallor and cyanosis. In certain instances a tumor in the region of the kidney is palpable. Their second case was treated with repeated injections of interrenin, the cortical hormone, and recovered.

IDIOPATHIC ENLARGEMENT OF THE HEART IN INFANCY AND CHILDHOOD

(Stoloff, *Amer. Jour. Dis. Children,* 1928, 36, 1204.)

He defines this condition as an enlargement of the entire organ or parts of it, which cannot be explained on a pathologic basis. In reviewing the literature he found 34 cases, reported as such, which were accompanied by pathologic reports. In five of these, anomalies or abnormalities of the heart precluded their consideration as instances of idiopathic enlargement. In another, the kidneys were of twice the normal size, which rules it out. In five of the remainder, histologic examination was not mentioned and in six more the microscopic examination revealed pathologic conditions in the musculature. There remain, therefore, only seventeen cases which can be considered as true idiopathic enlargement. These figures show the rarity of the condition. He reports another which was considered idiopathic until microscopic studies revealed lymphocytic infiltrations and myocardial degeneration generalized enough to explain the enlargement.

He calls attention to the slightness of the clinical manifestations with such extensive involvement of the heart. Most of the children were

*For record and address of author see "This Week's Issue," page 786.

said to have been well previous to the terminal illness, which was usually of short duration. Death was sudden and, as a rule, unexpected. Enlargement of the heart was noted clinically in only ten of the seventeen. Dyspnea, usually not marked, was not uncommon. Cyanosis of varying degrees was also frequently noted, but usually only immediately before death. Systolic murmurs were heard in five of the cases, but they were apparently functional, since necropsy did not reveal any organic cause for them. The presence of dyspnea in the absence of evidences of pulmonary or congenital heart disease should suggest idiopathic enlargement of the heart.

There is no doubt that idiopathic hypertrophy may be congenital, as it has been found in newborn infants. Whether it is always congenital is not known. It is conceivable that a congenital condition of this nature may lie dormant until an infection or some added strain may initiate or precipitate a failure of the heart.

ACIDOSIS AND ALKALOSIS

Although the diagnosis of acidosis is very commonly made, both by physicians and the laity, it is probable that the vast majority of those who make this diagnosis do not really understand what acidosis is, what the symptoms are, that there are different causes for it and that treatment must depend on the cause. Dr. Gamble, in the NEW ENGLAND JOURNAL OF MEDICINE, 1928, 199, 1318, has described the chemistry of acidosis and alkalosis in as simple language as is possible for such a complicated subject. It is worth reading and re-reading. Marriott and Hartmann in the *Journal of the American Medical Association*, 1928, 91, 1675, have approached the subject from a different point of view. Their paper is somewhat more difficult to understand. It emphasizes, however, and points out the difference in the etiology and, in consequence, in the treatment of the different forms of acidosis.

STATUS LYMPHATICUS

(Marine, *Archives of Pathology*, 1928, 5, 661.)

This article is a review of the present knowledge of status lymphaticus. It is impossible to abstract it in detail. He defines status lymphaticus as a constitutional defect, usually congenital, dependent on an inadequacy of some function of the suprarenal glands, the sex glands and the autonomic nervous system, and associated with lowered resistance to a great variety of nonspecific physical and chemical agents. Anatomically it is characterized by delayed involution or hyperplasia of the thymus, hypertrophy and hyperplasia of the lymph glands and lymphoid tissue of the various organs, under-development of the chromaffin, gonadal and cardiovascular systems and by certain peculiarities of external configuration.

The function of the thymus is unknown. There is no proof that it produces an internal secretion, but it seems to play some rôle in the maintenance of normal nutrition. The effect of removal of the thymus is unknown. All agree, however, that the thymus is an important source of blood lymphocytes and perhaps of eosinophiles. There is no known disease entity in which the thymus occupies the central or causal rôle. The lesions in status lymphaticus are distributed over the whole body and their relative importance cannot be evaluated. In it the thymus is usually larger than it should be, but there is no agreement as to its histology, which varies in different cases. The spleen is moderately enlarged, the malpighian bodies being prominent and the endothelial elements hyperplastic. The lymph nodes throughout the body are enlarged and there is hypertrophy of the oral and nasopharyngeal lymphoid tissues. The bone marrow often shows changes. The thyroid gland is enlarged. The suprarenals are small and the chromaffin tissue is reduced in amount. There are no significant changes in the sex glands. The cardiovascular system is underdeveloped and the blood shows a relative lymphocytosis. There are marked disturbances in the ossification of the bones.

There are no constant or characteristic clinical symptoms by which the condition can be diagnosed. The immediate cause of death is stoppage of the heart. He calls attention to the theory of MacLean and Sullivan that status lymphaticus is due to suprarenal insufficiency.

INFANT FEEDING AND DISEASES OF THE DIGESTIVE TRACT

The Curd and the Buffer in Infant Feeding

(Brennemann, *Journal of the American Medical Association*, 1929, 92, 364.)

Brennemann assumes that with evaporated milk there is an almost complete arrest of coagulation in the stomach. He argues that, if the addition of acid aids digestion simply by preventing coagulation in the stomach, the results should be as good when evaporated milk is given without acid as when acid is added; on the other hand, if the effect of the acid is due to the change of the hydrogen-ion concentration during digestion, evaporated milk with acid added should give better results than plain evaporated milk.

He, therefore, gave evaporated milk with and without added acid to a series of babies showing disturbances of digestion from parenteral infections. The same babies were given the milk with and without added acid at different times. He was unable to see any difference in the results obtained with the evaporated milk to which acid was added. Both worked equally well. The conclusion seems to be, therefore, that the improvement was due to the prevention of coagulation in the stomach and not to the change in the hydrogen-ion concentration.

The Effect on the Urine of Addition of Acids and Alkalies to the Diet of Infants

(Greene, *American Jour. Diseases of Children,* 1928, 35, 38.)

He studied the urinary changes in infants and in older children who received one of the following acids or alkalies in the milk ingested: tenth-normal and thirtieth-normal hydrochloric acid, lactic acid, lemon juice, sodium bicarbonate or sodium hydroxide in varying quantities and concentrations. His conclusions are that when hydrochloric acid in small amounts, 45 c.c. of tenth-normal solution, is added to a liter of milk, it is followed by the presence of casts and sometimes of red blood cells in the urine of infants. In older children, about 100 c.c. of this diluted acid is required to bring about similar irritative manifestations. When the acid is omitted, the pathologic urinary changes disappear in about 48 hours. The result was similar when 8 grams of calcium lactate were added to the milk. Lactic acid, when added in quantities of 8 c.c. to a liter of milk, only occasionally produces casts in the urine. Lemon juice in a concentration of 3% does not lead to any evidences of irritation.

Sodium bicarbonate, when given to infants and older children in large doses, 240 c.c. of a double normal solution, the equivalent of 152 grains daily, does not have the same irritating effects on the urinary tract that acid does. Sodium hydroxide, 240 c.c. of a tenth-normal solution in quantities of 100 c.c., does not produce the urinary changes noted when tenth-normal hydrochloric acid is added. The result is a slightly alkaline or slightly acid urine, which may account for the lack of irritative effects.

The addition of sodium hydroxide leads to the formation of the typical soapy stool. When the milk was skimmed the addition of sodium hydroxide rendered the urine markedly alkaline.

Self-selection of Diet by Newly-weaned Infants

(Davis, *American Journal Diseases of Children,* 1928, 36, 651.)

She allowed newly-weaned infants to choose their own foods in such quantities as they desired from a fairly wide range of commonly used natural food materials, unmixed, unseasoned and unaltered except, in the case of some, by cooking in the simplest manner. Her objects were to determine whether infants of weaning age could and would, when removed from the breast, choose their own foods from those placed before them, without aid, as do adults, and in sufficient quantities to maintain themselves. If they did, whether they would eat few or many of the large number of articles offered or eat indiscriminately what was nearest at hand, governed only by their caloric needs, or show definite preferences. If they did, would these preferences tend toward a vegetarian, a carnivorous or an omnivorous type of diet. Whether such infants would maintain themselves in a state of digestive health or would suffer impaired digestion with general discomfort, vomiting, diarrhea or undigested food in the stools, and whether their growth, eruption of teeth, gain in weight and general well being would equal those of infants fed in the usual way on the diets commonly prescribed for this age.

Two infants were studied for six months each and one for a year. They were able from the first to select their own foods from a list of simple, natural ones and in quantities sufficient to maintain themselves with apparently optimal digestive and good nutritional results. They were omnivorous in eating, were governed not only by their caloric needs, but showed definite preferences, which, however, changed from time to time and were unpredictable.

(Morse: These results are most disconcerting. It is difficult to know whether we ought to keep on with the time-honored methods of feeding babies, feed them all in this way, or combine the two. The latter seems most reasonable. Her results certainly seem to show that babies may be given a wider variety of food than has been the custom in the past. They do not show, however, that they do any better, if given it.)

The Etiology of Coeliac Disease: Preliminary Communication on the Blood Fat

(Moncrieff and Payne, *Archives of Disease in Childhood,* 1928, 3, 257.)

They define coeliac disease as a digestive disorder arising in infants and young children who show no evidence of chronic intestinal or other organic abdominal disease: it is characterized by a persistent malabsorption of dietetic fat which causes large stools containing an excess of split fat, enlargement of the abdomen, lack of growth and other symptoms. They state that the similarity between the stools of coeliac disease and those of abdominal tuberculosis, in which there is obstruction of the lacteals, suggests that coeliac disease is due to lacteal obstruction. They quote two cases in which the stools contained large amounts of fat. In the first, which was due to masses of tuberculous glands obstructing the lacteals, the blood fat was 0.145%, while in the other, in which the mesenteric glands were not enlarged and the intestine and pancreas were normal microscopically, the blood fat was 0.422%. They say that it is so common in speaking of coeliac disease to talk of the malabsorption of fat and to look upon the disease as essentially characterized by a failure of the intestine to absorb fat, that it might be expected that the fat in the blood would be considerably reduced below normal. They found in a small series of cases that the blood fat was markedly increased when the patients were not doing well and moderately increased when they were improving. They call attention to the facts that tests for pancreatic efficiency are normal in

coeliac disease, that the intestinal ferments have been shown to be normal and that the fat in the feces is normally split. They also mention the work of Bloor and his associates, which seems to show that the fat in the feces has no direct relationship to the fat in the food in many cases and represents rather some form of excretion from the intestinal tract. They conclude that the most probable source of the fecal fat seems to be excretion from the blood, either directly or indirectly, as is evidenced by the similarity of the fecal lipoids to those of the plasma. Moncrieff and Payne state that, if the conclusions of these authors are correct, a possible explanation of coeliac disease lies in the high blood fat associated with excretion in the feces of fat which has "leaked" from the blood. In other words, the primary fault in coeliac disease is not malabsorption of fat, but a malutilization of fat, a defect of fat metabolism possibly analogous to the defective utilization of sugar in diabetes. The high blood fat which they found in their cases is corroborative evidence.

The Treatment of Pylorospasm in Infants With the Roentgen Ray

(Wiener, *Jahrb. für Kinderheilkunde*, 1928, 122, 113.)

Wiener used the term pylorospasm as synonymous with pyloric stenosis. Like other Germans, he apparently does not distinguish between pyloric spasm and pyloric stenosis. Visible peristalsis is mentioned in four of the six cases which he treated and a palpable tumor in two. No mention is made of either peristalsis or tumor in the others. He gave one treatment in one case, two treatments in two cases, three treatments in one case, four in one, and five in one. His results, as given, were most remarkable. Vomiting diminished at once and soon ceased, and the bowels, which were constipated, soon began to move regularly. A gain in weight was marked in all and in several began almost immediately. He claims that relief is obtained more quickly in this way than in any other and that the results can often be obtained without changing the character of the feeding. He says, as of course is true, that if cure can be obtained in this way without surgical interference, it is a great therapeutic advance.

SUNSHINE, ULTRAVIOLET IRRADIATION AND VITAMIN D

(Morse: The number of articles which have appeared during the last year on sunshine, ultraviolet irradiation, Vitamin D and the activation of various foodstuffs by irradiation in the newspapers, lay journals and medical journals is so large that it is impossible to abstract them. It is also very difficult to evaluate them, because so much bunk is being written about these matters by people who are ignorant or badly informed and because the suspicion is constantly aroused that many of these articles are inspired or paid for by those who have an interest in the sale of various types of window glass, lamps and food. The conclusions reached, moreover, in those articles which are apparently written by unbiased and unprejudiced observers as the result of controlled and scientific study, are so different that it is hard to tell what is true and what is not. Only a few of the articles, which seem most important, are, therefore, mentioned.)

Seasonal Variation of the Antirachitic Effect of Sunshine

(Tisdall and Brown, *American Journal of Diseases of Children*, 1928, 36, 734.)

As the result of a study of the antirachitic effect of the sun's rays throughout a period of one year at Toronto, they conclude that the sun's rays during the latter part of October, all of November, December, January, and the first part of February in the latitude of Toronto, produce a slow, but definite, antirachitic effect on rats fed on a rachitogenic diet. A sharp increase occurs in the antirachitic effect of sunshine about February 15. This increased antirachitic effect of sunshine continues throughout the latter part of February, all of March, April, May, June, July and August, September and the first half of October. The antirachitic effect of sunshine during April, May, June, July, and August is approximately eight times as great as during November, December and January. The marked decrease in the antirachitic effect of sunshine occurs about October 15.

The Penetration of Ultraviolet Rays into Live Animal Tissues

(Macht, Anderson and Bell, *Journal American Medical Association*, 1928, 90, 161.)

(Morse: This article comes from the Pharmacological Research Laboratory of Hynson, Westcott & Dunning and the Physics Research Laboratory of the Hanovia Chemical and Manufacturing Company.)

They first call attention to past observations showing that the depth of penetration for the shorter ultraviolet rays into the skin is for the most part not more than 0.1 mm. They also call attention to the fact that the experiments of the older authors were all performed with dead skin. In these studies the photodynamic effects were produced in live animals. They claim to have distinctly established by their experiments that the penetration of ultraviolet rays through the living skin and other tissues is much greater than has hitherto been supposed. Some of the shorter ultraviolet rays penetrate more deeply than the longer. A marked difference was noted between living and dead skin. White, human skin is more permeable to ultraviolet irradiations than negro skin on account of the presence of pigment in the negro skin.

The Action of Ultraviolet Irradiation on the Bactericidal Property of the Blood

(Gonce and Kassowitz, *Journal of the American Medical Association,* 1928, 90, 280.)

They say that the killing power of blood *in vitro* for certain test bacteria before and after general irradiation offers one criterion for the changes in the antibactericidal properties that have taken place in the body. They found that continuous, daily, quartz lamp irradiations for more than four weeks with routine exposure and distance lead, in the majority of all cases examined, to a lowered bactericidal property of the blood. Completely normal persons, free from any infection, were apt to show improved bactericidal property after continuous ultraviolet irradiations; those suffering from chronic infections and postinfectious conditions after daily quartz lamp treatments were apt to show a drop in the bactericidal property of the blood. The increased or reduced bactericidal property of the blood after ultraviolet irradiation seems to depend largely on the leucocytic reaction of the body. An increased number of white cells, especially of polymorphonuclears, is associated with improved killing power for cocci. Two different individuals may, however, show the same number of leucocytes and the opposite antibacterial response. It is necessary, therefore, to assume differences in the leucocytic ability of different individuals.

The Effect of Carbon Arc Irradiation on the Health of a Group of Infants

(Barenberg and Lewis, *Journal of the American Medical Association,* 1928, 90, 504.)

The object of their investigation was to determine primarily whether ultraviolet radiations diminished the occurrence of respiratory infections. They found that, in spite of systematic exposures to the radiations of the carbon arc lamp, no comparative diminution in the incidence of respiratory infections was noted in a series of infants treated for a period of three months. Growth in weight and height during the first 6 weeks was greater in the irradiated than in the non-irradiated group of infants. During the second 6 weeks, however, growth was less among the irradiated infants. The number of erythrocytes and leucocytes did not seem to be affected by irradiation. The hemoglobin percentage of the irradiated group was maintained during the first period, whereas a fall of 12% was noted in the non-irradiated.

The Prevention of Colds by Ultraviolet Irradiations

(Editorial, *Journal of the American Medical Association,* 1928, 90, 547.)

In this editorial the literature of this subject is briefly reviewed. Attention is called to the difference in the results claimed and also to the difference in the character of the investigations. The conclusion is that "thus far, apparently, the family physician charged with keeping his patients well is in a dilemma. On the one hand are well controlled experiments with negative results in which the dose was large. On the other hand are imperfectly controlled experiments the success of which the investigators attribute to low dosage. Positive statements, faith and investments may well await further evidence."

Quartz Lamp Therapy in Human Rickets and Rachitic Spasmophilia

(Gerstenberger and Hartman, *Journal of the American Medical Association,* 1929, 92, 367.)

They found that weekly exposures to the ultraviolet rays produced by the quartz lamp, in doses of one erythema unit for colored infants and less for white infants to both front and back, brought about healing in practically the same time required by the heavier schedules at present generally used. Complete healing occurred in from five to eleven weeks, according to the type of rickets present.

(Morse: These results, if generally accepted, will seriously interfere with the office practice of many pediatricians.)

Irradiated Ergosterol

Numerous papers on irradiated ergosterol have appeared within the last year and a half. As is well-known, it has been learned that it is ergosterol, not cholesterol, which is activated by the ultraviolet rays. It has been proven conclusively that activated ergosterol is a highly potent antirachitic both for animals and for man. It is roughly 2000 times as potent as activated cholesterol and 100,000 times as potent as cod liver oil. One ounce of average activated ergosterol is the equivalent of from 2 to 6 tons of cod liver oil. It is able to prevent or cure rickets in rats in daily doses of 1/2000 milligram or less, according to the purity of the preparation. It is estimated that for a baby growing at the normal rate a daily dose of 0.5 milligram is sufficient to prevent rickets and that a daily dose of about 2.5 milligrams is sufficient to cure it. The potency of different preparations of irradiated ergosterol varies materially, however. The clinical advantages of ergosterol over cod liver oil and ultraviolet irradiation are evident, provided the potency of the individual specimen is known.

Unfortunately, however, overdosage with activated ergosterol may cause serious and even fatal symptoms. Animals become somnolent, lose their appetite, become cachetic and die. Post mortem, large calcium deposits are found throughout the organs, particularly in the vessel walls, heart muscle, stomach wall, lungs, kidneys and intercostal muscles. The kidneys and adrenals are shrunken, the spleen atrophic

and the mucous membrane of the intestines hemorrhagic and ulcerated. So far as I know, no serious results have been reported in infants. The doses which caused trouble in animals were very large, however. The difference between a toxic and a healing dose is very large. In rats, for example, a healing dose is 1/10,000 of a milligram, while the toxic dose is greater than 1 milligram. It seems rather improbable, therefore, that babies and children would be given large enough doses to cause serious trouble. Nevertheless, it is probable that the amounts now recommended and given to infants are larger than necessary and may lead to hypermineralization. Until the activity of a given specimen of irradiated ergosterol can be more acurately estimated than at present and until the dosage of a standard preparation has been more definitely fixed, it seems advisable for the average practitioner to keep on with cod liver oil and sunshine and leave the various preparations of irradiated ergosterol alone. This is also the feeling of the Council on Pharmacy and Chemistry of the American Medical Association which has requested one of the reliable manufacturers of this country to withdraw its product for the present.

Hess and Lewis, *Journal of the American Medical Association,* 1928, 91, 783.

Pfannenstiel, *Klin. Wchnschr.,* 1927, 46, 2310.

Kreitmar and Moll, *München. med. Wchnschr.,* 1928, 75, 637.

Greene and Mellanby, *British Medical Journal,* 1928, 2, 691.

Klein, *Journal of the American Medical Association,* 1929, 92, 621.

Abstracts, *American Journal Diseases of Children,* 1928, 36, 1044, 1045.

Discussion, *Jahrb. für Kinderheilkunde,* 1928, 122, 242.

DISEASES OF THE LUNGS

Peribronchial Infiltration after Respiratory Infections in Childhood—A Clinical Syndrome

(Heiman and Cohen, *Archives of Pediatrics,* 1927, 44, 677.)

This syndrome consists of dulness on percussion, usually over both lower lobes, often in the axillary regions, low grade fever for a long period, from 100°F. to 101°F. as a rule, and, in most cases, cough. Râles, bronchial breathing and changes in the voice are conspicuous by their absence. Roentgenograms show fine infiltrations in about half of the cases with accentuation of the lung markings in most of the others. Although the course tends to be chronic, the pulmonary involvement may be found early in the course of the primary disease. It thus serves to prolong the higher fever and toxemia of the initial infection. In the chronic stages the patient suffers little, if the fever is not high, but, at times, the cough is distressing. They believe this syndrome is a streptococcus pneumonia of the interstitial type, without alveolar exudation. They state that this form of pneumonia is not uncommon in children. The interstitial lesions may completely resolve after a long time, or become indurated, causing a permanent dulness on percussion of the region affected. The prognosis is excellent. There were no deaths in their cases and no children suffered from deterioration of general health.

(Morse: Our conception of pneumonia in early life has changed very materially within the last few years. It is now believed that many of the conditions which are commonly described as bronchopneumonia are really mainly interstitial in distribution. It is probable that the authors are describing a mild type or the results of this interstitial type of "bronchopneumonia" in early life.)

Differential Diagnosis of the Pneumonias in Childhood

(Smith and Langmann, *Transactions of the American Pediatric Society,* 1928, 40, 42.)

They say that lobar pneumonia occurs in infants and young children more commonly than any other pneumonia and exactly resembles that seen in adults and older children. Bronchopneumonia is a term used to cover several different entities which fall into certain definite groups.

1. *Peribronchial Pneumonia or True Bronchopneumonia.* The lesion is an infiltration with lymphocytes of the submucosa of the smaller bronchi. A patchy consolidation of some adjacent alveoli is also found. The exudate is poor in fibrin. There are many collapsed alveoli. Other alveoli are dilated. Resonant râles are the characteristic physical signs, but there may be signs of consolidation. The Roentgenogram does not show a shadow, even when there are physical signs of massive consolidation and a pseudolobar involvement at autopsy. The organism found is usually a streptococcus and the mortality is high.

2. *Lobular Pneumonia.* The lesion is an acute exudate into some of the lobules of the lungs, as if the infection had traversed the lumen of the bronchi and entered the alveoli by this route. There is a patchy consolidation. The exudate contains considerable fibrin, many leucocytes and often many red cells. The physical signs are resonant râles or signs of consolidation over the involved lobes. The Roentgenographic shadow varies. In some cases it appears as a patchy or mottled area, which spreads to a diffuse shadow, but still is not homogeneous. At times a shadow indistinguishable from lobar pneumonia may appear at some stage. The bacteriology is varied. Streptococci, pneumococci and influenza bacilli are found.

3. *Interstitial Pneumonia.* The characteristic of interstitial pneumonia is an enormous distension of the lymphatics of the interlobular

connective tissue septums by large numbers of leucocytes. The pleura is usually involved. The lung gives physical signs of consolidation. Microscopically, there is little, if any, exudation into the alveoli, but merely collapse and acute congestion. A hemolytic streptococcus is found.

4. *Mixed Forms.* The finding of mixed forms confuses the matter and is responsible for the lack of clearness in descriptions in textbooks. Many cases are definitely lobular, but there is a peri-bronchial pneumonia elsewhere in the lung. There may also be an acute interstitial lesion.

ANEMIA IN INFANCY AND CHILDHOOD

In a symposium before the Section of Pharmacology and Therapeutics of the American Medical Association at its meeting in June, 1928, Whipple (*Journal of the American Medical Association*, 1928, 91, 863) calls attention to the danger of drawing deductions from one type of anemia and applying them to another. He also says that the reactions in animal experiments may give suggestions as to procedure in the treatment of human anemia, but subsequent observations must be careful and long continued on the human cases to establish similarities or differences in the clinical reaction as compared with the experimental. He notes that in experimental anemia in animals iron is inert in the short experiments, but distinctly potent in the long period anemias. The most potent food for blood hemoglobin regeneration in simple anemia is liver. Kidney is practically on a par with liver. Apricots and peaches are also quite potent. Large or small doses of iron salts may cause an increased production of blood hemoglobin, but a continuing diet period of similar iron feeding plus a diet of liver or kidney will show the sum of the two expected reactions—the reaction of the iron feeding plus the reaction expected from the liver or kidney diet.

Human anemias compared with the relatively simple experimental anemias are usually difficult and complex. Anemia associated with infection is little understood and calls for much careful study. Dietary anemias are probably much more common than is usually appreciated. Such anemias are not uncommon in children and probably are usually due to an overwhelming preponderance of the dairy products in the diet. A limited diet of milk products promptly causes anemia in pups and rats. He believes that pernicious anemia belongs in the group of dietary anemias. He calls attention to the fact that bone marrow abnormalities clear up promptly on administration of a liver diet. It is very difficult to correlate this fact with a progressive intoxication. It favors the assumption that pernicious anemia is a deficiency disease.

Simple anemia in animals can be controlled at will by diet factors and the evidence goes to show that simple anemia in human beings can likewise be readily controlled by diet therapy. The anemias complicated by infection call for much more study, but all diet factors and favorable inorganic salts should be carefully tested in these complicated human anemias. He calls attention to the fact that the combination of liver extract and liver is more potent than the liver extract alone. He thinks that it may be of some value in the liver extract therapy of secondary anemia. (Morse: Various authors have found that liver extract was quite potent in the secondary anemia of rats caused by a limited milk diet.)

Means and Richardson (*Journal of the American Medical Association*, 1928, 91, 923), in discussing Minot's discovery of the value of liver in the treatment of pernicious anemia, call attention to the fact that Whipple's results would lead to the expectation that liver feeding, if effective at all in human anemia, would be effective in the secondary type. Nevertheless, Minot found that it was a specific in the pernicious type. They state that pernicious anemia is due to a shortage of some specific substance which is responsible for the abnormalities in the marrow and in the blood. The cause of the shortage is not completely known. Neither is it known whether the normal human being requires any extraneous supply of this specific substance or can synthesize what he needs. Liver and kidneys contain large amounts of this specific substance.

The course, the characteristics of the blood and the pathologic changes in pernicious anemia are so different from those of simple anemia and secondary anemia that it does not seem as if the latter could be due to a lack of this specific substance. Furthermore, in most simple and secondary anemias, especially in infancy and childhood, the cause is obvious. In certain types, however, the cause is not evident, the changes in the blood resemble, to a certain extent, those in pernicious anemia and the changes in the bone marrow are sometimes similar. It would seem reasonable, therefore, to hope that in these cases liver might be beneficial. In the ordinary case of simple anemia or secondary anemia liver could hardly be expected to do any good. Nevertheless, Whipple's observations on the treatment of simple anemia in animals with liver and his observation that the combination of liver and iron was effective, suggest that it may.

(Morse: Various papers have appeared on the treatment of anemia in early life with liver or liver extract. The observations in most of them, however, have been poorly controlled and are inconclusive. Most observers have found liver and liver extracts of but little use in secondary anemias from whatever cause, but brilliant results have been reported in occasional cases.)

Faber (*American Journal Diseases of Children*, 1928, 36, 1121) calls attention to the difficulty in the diagnosis of pernicious anemia in infancy, but says, in discussing several of the re-

ported cases, that the blood pictures in some of them show as a common feature the defective erythroblastic maturation in the bone marrow, which Peabody has recently described as occurring so constantly in pernicious anemia. Liver and liver extract are known to promote maturation and, therefore, should be of use in anemia showing this picture. He reports three cases—two in infants and one in a boy of 4½ years, which were studied most carefully. The blood picture in all resembled that of primary anemia. There was a striking clinical and hematologic improvement associated with a characteristic transient rise in the circulating reticulocytes following the administration of liver extract in the infants, but none in the boy. He thinks that one of the patients, aged 9½ months, probably had true pernicious anemia, as shown by the blood picture, achlorhydria and the typical response to the administration of liver extract. He concludes that at any rate there appears to be no doubt that as early as the sixth week of human life a type of anemia does occur in which the maturation of erythrocytes in the bone marrow is defective or inadequate and in which liver extract is of striking therapeutic value. (Morse: The success which Faber and a few others have obtained in the treatment of anemia in early life in a few instances does not justify us, however, in giving liver and liver extracts to every poor baby or young child that is pale or whose parents think it is anemic. Neither does it justify us in feeding liver to babies who ought to be taking breast milk. Liver and liver extract should, however, be carefully and thoroughly tried out in the treatment of, at any rate, the severe anemias in both infancy and childhood by men competent to do so and in institutions where the results can be carefully evaluated.)

Mackay (*Archives of Disease in Childhood*, 1928, 3, 117) studied 541 infants under 2 years of age in London. She found that the great majority of artificially-fed infants and many breast-fed infants are anemic. Treatment with the mercury vapor quartz lamp had no influence in preventing or curing this anemia and no definite relation was shown between the seasonal intensity of ultraviolet light and the incidence of anemia. Out-door life did not cure it. Anemia develops in infants on many types of diet, and no evidence was obtained to associate it with vitamin deficiency or with fat toxemia. This prevalent anemia is due to iron deficiency, perhaps with some associated factor, and shows itself from about four months of age.

It can be prevented or cured by the administration of a soluble iron salt by mouth. Iron can be satisfactorily and successfully administered in the form of iron and ammonium citrate, contained in a dried milk powder, or added in solution to the daily foods. Iron should be given to artificially-fed infants from the first months of life, as the hemoglobin can thereby be maintained at a higher level than if it is given later. Its early administration is particularly important in babies who are small at birth. Artificially-fed infants receiving such iron show a striking improvement in general health and resistance to infection. The incidence of diseases of the respiratory tract, as assessed by the author, was, in the iron group, only half that in the control group.

THE MONOCYTE IN ACTIVE TUBERCULOSIS

(Blackfan and Diamond, *American Journal Diseases of Children*, 1929, 37, 233.)

They studied the blood of seventeen infants and children, proved to have or suspected of having tuberculosis, by the supravital staining technic. They review the work of Cunningham and his co-workers on the rôle of the monocyte in tuberculosis in animals and in human tuberculosis. Their conclusions are: the degree of activity in tuberculosis is reflected in the peripheral blood of infants and of children. A high monocyte-lymphocyte ratio and the presence of epithelioid cells usually mean an active tuberculous infection. An increasing monocyte-lymphocyte ratio and the persistence of a high absolute monocyte count signify extension of the process. Reversal of the ratio (a fall of monocytes and an increase in lymphocytes) denotes healing lesions. In cases in which the diagnosis is questionable, a study of the blood by the supravital technic may lead to a correct diagnosis. When the diagnosis is established, supravital studies of the blood may be of prognostic value in indicating whether the lesion is progressive or undergoing regression.

(Morse: This work is most interesting and bids fair to be of great assistance not only in the diagnosis of obscure cases of tuberculosis but also in prognosis and as an aid to treatment. It is unfortunate that the procedure of supravital staining is so difficult that this method is hardly feasible for the general practitioner. It is to be hoped that some other way of recognizing these cells in fixed smears may be discovered.)

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 15151

TWO WEEKS' COUGH AND VOMITING

MEDICAL DEPARTMENT

A married American woman twenty-two years old entered May 25 for study of a chest condition. The chief complaints were vomiting and cough of two weeks' duration.

January 21, four months before admission, she was normally delivered of her second child. A report from the hospital states that the child developed gonorrheal infection of the eyes and that the mother said her first child, born two years earlier, had the same thing. There were no complicating factors at the second delivery except that she lost, the doctor told her, a great deal of blood. On returning home two weeks later she was so weak that she was unequal to any exertion and felt tired out all the time. In two months she had regained a good deal of strength. Then she began to have afternoon headaches. Three weeks before admission she worked very hard in moving her household goods and also got wet through in a rain. She developed a hard hacking cough, unproductive at first, but so severe that the paroxysms bent her double and left her exhausted. She began to have nausea and vomiting and could keep nothing down. She called a doctor, who found no temperature, lungs normal, and put her to bed. She stayed there three days, then got up, feeling no better. Two weeks before admission while working one evening she suddenly felt weak and nauseated. She had a prickling sensation in her arms and hands, everything turned black before her eyes and she fell unconscious. Her husband found her vomiting, very pale, cold, trembling, cyanotic about the lips, with twitching of the muscles and very rapid heart. The following day she was very weak and half asleep all the time. Following this her cough became less hacking and produced small amounts of salmon colored sputum. She now had somewhat less vomiting and could usually retain one meal a day. She continued very weak and often had mild sweats. At one of his visits the doctor said her pulse was 130 but her temperature subnormal. A week before admission she began to have a rattle in her throat which made her respirations very noisy. She could however stop this by coughing up sputum. Her cough was less severe and less paroxysmal. She noticed that she had lost about fifteen pounds in three weeks. Six days before admission she coughed up some blood streaked sputum. She began to be unable to sleep because of orthopnea. She had a sensation of a lump in her throat, relieved by sitting up. Since that time she got what little sleep she had sitting in a chair. Four days before admission a consulting physician said she had some water on the right lung.

Her family history shows heart trouble in a grandfather and the death of an uncle from tuberculosis.

Twelve years before admission she had a slight illness which was called appendicitis. Two years later she had a mild case of influenza. Two years before admission, in the fifth month of her first pregnancy, she was in a hospital for study of abdominal pain thought possibly to be appendicitis. Before the birth of her first child, four months later, she had nosebleeds and began to urinate once at night. This had continued ever since. Two urine examinations in the hospital showed a slight trace of albumin and a few leukocytes. A third specimen on the day of discharge showed complete reduction for sugar; a fourth showed no reduction. The blood pressure at admission was 140/70 and at discharge 130/75. She reëntered the hospital and was normally delivered. The hospital report does not mention gonorrheal infection of the eyes. The patient denied lues and Neisser infection by name and all symptoms. Since her first delivery she had had slight white vaginal discharge. A year before admission she weighed 117 pounds, her best weight.

Clinical examination showed a fairly well developed, poorly nourished girl with pale skin and slightly cyanotic lips. Chest symmetrical, but expansion less on the right. Signs of a small amount of fluid at the right base. Right back dull, with medium and coarse râles at the scapular region. Left back slightly dull at the scapular region, with increased fremitus and medium moist and crackling râles. Heart normal except for a low, short systolic murmur at the apex. Blood pressure 190/140 to 112/65. Abdomen negative. Pelvic examination showed some procidentia and retroversion, with lacerated cervix and some tenderness in the vaults.

Urine: amount normal, specific gravity 1.014 to 1.003 (at a two-hourly test 1.005 to 1.012), a slight trace to a large trace of albumin at all but one of 19 examinations; two catheter specimens showed occasional leukocytes and epithelial cells, one showed rare red cells. Renal function: two tests 0, one 5 per cent. Cultures of the two catheter specimens both showed no growth. Blood: 7,200 to 6,000 leukocytes, polymorphonuclears 64 per cent, hemoglobin 60 to 70 per cent, reds 3,500,000 to 4,300,000, slight anisocytosis, poikilocytosis and achromia, platelets normal. Non-protein nitrogen May 26, 80, increasing steadily to 270 July 12. Creatinin

5.7, increasing to 15.2. Uric acid 7.4 to 11.5. Phosphorus 8.6 to 16.2. Hinton negative. Sputum: no blood or tubercle bacilli at four examinations. Stools: guaiac strongly positive at one of four examinations, questionably positive once. Urethral and cervical smears showed no Gram-negative diplococci.

X-ray examination. The left diaphragm was distinctly seen, sharp in outline. There was some mottled dullness extending outward from the left hilus. The right diaphragm could not be made out. There was decreased radiability of the lower two-thirds of the right lung field. The border of the heart was distinctly seen. The right apex appeared normal. Another examination three days later confirmed the previous findings. The costophrenic sinus on the right was obliterated. There was mottled dullness at both lung roots, more on the right. On this side it extended outward and downward along the course of the bronchi. The interlobar septum was visible on the right. All the lung markings were thickened and beaded. The upper portion of the lung fields was clear.

Temperature normal until May 30, then elevated for five days, reaching 102°, afterwards normal except for a slight elevation to 99.6° June 13 to 15 and a rise to 102°-99.7° three days before death. Pulse 70 to 128. Respirations normal.

May 27 examination showed a shower of medium crackling râles in the left midscapular region. May 31 there were signs of consolidation in the right midlobe and part of the lower lobe, possibly with a small amount of fluid. The following day in addition to nausea and gastric instability she complained of pleural pains along the lower sides of both chests. The night of June 3 she had an acute episode with weakness and syncope accompanied by vomiting and a very feeble pulse. The heart was rapid and regular but showed gallop rhythm. The process in the chest cleared slowly and in two weeks was practically gone. June 9 there was some edema of the face and abdomen. June 15 the blood showed a marked increase in phosphorus (13.3), creatinin (9.5) and non-protein nitrogen (140). She had some pain and redness of the smaller joints. X-ray examination June 18 showed the heart shadow prominent in the region of the left ventricle. June 22 it was thought that a rough localized pericardial friction rub was heard. There was some twitching of the muscles. The patient at no time had a true convulsion. She became increasingly drowsy. The hemoglobin fell to 35, the red count to 1,875,000. A cellulitis developed over the right buttock. The leukocyte count was 17,200. The gums oozed a little blood. July 15 the patient died.

Discussion

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

"A chest condition" is medical slang. It is not English or any other language. The fact that we are used to those terms in our records and in our talk does not justify us. It would be far better to say the study of a chest lesion or a disease of the chest. "A chest condition" does not mean anything.

Two weeks before admission she had a fainting fit with peripheral anemia which made her have these sensations in her hands, and anemia of the retina whereby she had black sensations before her eyes.

The rattle in her throat stopped by coughing up sputum is not significant.

What can we get from that history? I do not think we have much until the last line. She had some disease which produces cough, dyspnea, orthopnea, fever, weakness and sweating following a confinement. She had, then, an infection, I suppose above the diaphragm. Whether it was in the lung as well as in the pleura I do not think we can yet decide.

That patient had about the average family history. Most of us have as much as that.

I get nothing out of that past history. We have to use the present illness in evaluating the clinical examination.

NOTES ON THE PHYSICAL EXAMINATION

The low specific gravity of the urine seems important. Judging by the number of examinations they were interested in the urine in this case.

The blood shows a secondary anemia.

That non-protein nitrogen is a convincing figure. There is no doubt about the meaning of that on July 12.

Of course "pleural pains" is not a proper term. They ought to say "pain increased with inspiration". We cannot rightly make the diagnosis of pleural pain.

DIFFERENTIAL DIAGNOSIS

We ought to come out right on this case. We have a history which does not in any way suggest kidney disease except for the presence of headaches, a little more frequent than a woman has a right to have after a normal delivery. There are no other helpful data until we come to the urine examination. Chronic nephritis is strongly suggested. There is nothing so far that I can see to make us think of anything else. The laity are always looking for pain in the back, but we do not expect to find evidence of nephritis until there are heart symptoms or brain symptoms or until we examine the urine. Examination of the urine shows evidence of a practically complete destruction of kidney function. The blood retention products confirm that.

At the end we see what we ordinarily call uremia. Secondary anemia increasing under observation is just what we expect. Chronic nephritis, then, is the underlying and main disease in this case. Then we have a variety of infections. I suppose she had bronchopneumonia and pleurisy on the right, then possibly a pericarditis, certainly a cellulitis of the buttock and also suggestions of an arthritis of the type that we get with sepsis elsewhere.

What type of nephritis ought to be found? We have not much to guide us. But at her age a *chronic glomerulonephritis* is commoner than any other kind. Merely on account of age I should say that is the probable diagnosis.

Why did she not have a high blood pressure?* I do not know. She ought to have. I cannot account for that; but it is not in any way unprecedented. In each series of well studied kidney cases there is a certain respectable but unexplained minority that do not have high blood pressure. Absence of that is not enough to make us withdraw our diagnosis of chronic nephritis.

From the X-ray the heart seems to be large, and I believe the left ventricular wall to be hypertrophied. The right pleura should be obliterated partially or totally from the pleurisy which we have every reason to believe she has had. There is no evidence in the lung of the bronchopneumonia which it seems probable that she had. But we ought not to see anything of it at a necropsy so long after the resolution of such a process.

A STUDENT: Is there any possibility of her arthritis and her lesion in the heart being caused by gonococcus septicemia?

DR. CABOT: Gonococcus septicemia does not seem to me at all probable. In the first place it is a rare disease. Dr. W. S. Thayer of Baltimore has collected an extraordinarily large group of cases; but I have hardly ever seen it here, although for a long time we were looking for it on account of the interest of Dr. J. H. Wright in that subject. The fact that the child had gonorrhea in her eyes does not in any way suggest that it had gone beyond the pelvic organs from which the child got it. It does attack the heart; but gonococcus endocarditis is very rare. I never make that diagnosis without a blood culture. We have not much to indicate heart disease in this case; and the joint symptoms, such as they are, are not in any way suggestive of the commonest joint symptoms of gonorrhea. Those symptoms are stiffness and pain which last through months, usually in a few joints, more often in an ankle or a wrist than anywhere else.

A STUDENT: What are the possibilities of heart disease in a case of nephritis without hypertension like this?

*The blood pressure was omitted in the summary as Dr. Cabot had it.

DR. CABOT: That is of hypertrophied heart?

A STUDENT: Yes.

DR. CABOT: They are good, I think. I expect to see the state of things which has so often been found at our post mortems here,—chronic nephritis, big heart. As to her low blood pressure, we have to say that we do not know.

A STUDENT: What was her blood pressure when she was here?

MISS PAINTER: 190/140.

DR. CABOT: I see. Then that is perfectly orthodox.

A STUDENT: Do you expect such a low red count in cases of this kind?

DR. CABOT: It is not at all uncommon in cases of chronic nephritis. I do not mean that it is the usual count. That is about three million. But this is not much lower. I have seen a good many lower than this.

A STUDENT: What causes it? What becomes of the red cells?

DR. CABOT: I do not know.

A STUDENT: How do you explain the rapid sequence of the thing?

DR. CABOT: From the chronic nephritis, very likely helped on by the anemia.

A STUDENT: How do you account for the fainting?

DR. CABOT: Many normal people faint, and any person with a chronic nephritis or anemia is especially likely to faint.

A STUDENT: Would you include her pericarditis as a part of the chronic nephritis?

DR. CABOT: Yes. In chronic nephritis perhaps the commonest terminal infection, certainly as far as I remember, is pericarditis.

A STUDENT: Would you call a nephritis coming on so rapidly a chronic nephritis?

DR. CABOT: Yes. It is only the *symptoms* that come on rapidly. In many cases we know that this disease can be compared to floating ice. Most of it is submerged. If this patient had been having yearly examinations of urine the nephritis would have shown up years before this.

A STUDENT: Do you think there is evidence of acute rheumatic fever?

DR. CABOT: There is not enough. We have to have more.

A STUDENT: Is there any indication of a chronic process in the lung?

DR. CABOT: What indication is there?

A STUDENT: She had a cough for a long period of time.

DR. CABOT: They said that the signs cleared up. Nothing much is said about it at the end.

A STUDENT: There were signs of consolidation and of fluid in the chest, and her temperature was elevated for five days. What were the signs in the chest due to?

DR. CABOT: They may have been due to edema fluid alone, with condensation of the lung above the fluid, without bronchopneumonia. It is per-

fectly possible that that is all she had. But with the presence of the râles it seems more like bronchopneumonia. As to why she did not have temperature I have nothing in particular to say.

A STUDENT: How about the wide range in the specific gravity of the urine?

DR. CABOT: I do not call it wide. Seven is not much.

A STUDENT: Is there any connection between chronic nephritis and the headaches?

DR. CABOT: They usually come in the nephritic attacks.

A STUDENT: Do they characteristically come in the evening?

DR. CABOT: No; usually in the morning.

A STUDENT: Were her eye grounds examined?

MISS PAINTER: No, they were not.

A STUDENT: What is the significance of the blood in the sputum?

DR. CABOT: Blood streaks mean something very different from blood in quantity. In general you find that in big hospitals where they are taking histories for tuberculosis they do not pay any attention to streaks of blood. Blood in teaspoonful quantities they think is important.

INTERPRETATION OF X-RAY

The appearance is that of a bronchopneumonia process, most marked around the right descending bronchus.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Chronic nephritis.
Uremia.

DR. RICHARD C. CABOT'S DIAGNOSIS

Chronic nephritis, probably glomerular.
Secondary anemia.
Bronchopneumonia and pleurisy, right.
Cellulitis of the buttock.
Possibly acute pericarditis.
Hypertrophy of the left ventricular wall.

ANATOMIC DIAGNOSES

Chronic glomerulonephritis.
Cellulitis of right thigh.
Atheromatosis.
Slight cardiac hypertrophy.
Fibrinous pericarditis.
Acute colitis.

DR. TRACY B. MALLORY: This patient did show a chronic glomerulonephritis. The kidneys were considerably atrophied, the combined weight of the two being only 130 grams. The capsules stripped with considerable difficulty, leaving coarsely granular, grayish surfaces. The cortex was so narrowed as to be barely discernible above the base of the pyramids. She had an anomaly of the pelvis and ureters on both sides in the form of a marked narrowing or stricture of the pelvis just above its juncture with the ureter. I do not believe however that it had anything to do with her symptoms or the development of the renal lesion. The process was symmetrical on two sides, and in all probability was a congenital anomaly. Strictures of congenital origin are sometimes found at that spot where the pelvis of the kidney joins the ureter.

The heart was very slightly hypertrophied. There was a slight degree of fibrinous pericarditis, and a slight colitis of the type that we usually see in uremia.

Microscopic examination of the kidneys showed the vast majority of all the glomeruli completely sclerosed. The process was evidently so old that there were very few traces of the original stages left. The only acute lesions present in the kidneys at all were vascular ones.

She had also a quite acute type of atheromatous deposit in the aorta. The entire intima was studded with minute yellow plaques. I think that perhaps the progression in this case is to be explained by a vascular process superimposed upon a very old chronic glomerulonephritis. There seems to be no evidence that the glomerular lesions as such had been progressing recently.

The other important finding and the immediate cause of death was a cellulitis of the right thigh.

A STUDENT: What of the lungs?

DR. MALLORY: They were perfectly negative.

DR. CABOT: I want to speak briefly of a chronic case that I have had something to do with lately and on which I made a mistake which I want you all to avoid. We all learn by our mistakes. This case was of a lady about fifty-five whom I saw six years ago with a large abdominal tumor on the right side which one of our surgeons here operated upon and removed. It was a hypernephroma. There was no evidence at the time of operation of any metastasis or of anything at all wrong with her except for this hypernephroma, which was bigger than a Rugby football. She was perfectly well in all respects for three years after this. She went abroad and was very active last summer. This autumn she began to have severe pain in the right back. Before that pain she had dragging and indefinite symptoms in the upper right abdomen for two months or more. She as well as her doctors knew she had had hypernephroma and that there was likelihood of metastasis, and could not help thinking of what this might mean. The pain continued and increased in severity until she could not go twenty-four hours without half a grain of morphia. The pain extended around the waist to the midline and to the right leg. She was examined by the surgeon who operated upon her, later by an orthopedist and by a first rate neurologist. None of them could find anything wrong or could make a diagnosis. We now know the diagnosis, and what we all should have

thought of we did not think of. Of course we all thought of recurrent malignant disease and were scared. We all feared that she had recurrence of this tumor in the bone, presumably in the spine, and were pretty sure she would have to have an awful life and die. She is now rapidly getting well. What I want to bring out is the thing of which none of us thought.

A STUDENT: Gall bladder disease?

DR. CABOT: I cannot say that she has not got that besides, but gall bladder disease could not cause the pain she had.

A STUDENT: Floating kidney?

DR. CABOT: No. Certainly first rate surgeons could not feel any.

A STUDENT: Subdiaphragmatic pleurisy?

DR. CABOT: I do not know any record of such pain from it.

A STUDENT: Renal calculus?

DR. CABOT: Everything was carefully done. I cannot name a method of examination that we did not try, and there was no evidence of stone.

A STUDENT: How about an extra rib?

DR. CABOT: It was not there.

A STUDENT: Ovarian tumor?

DR. CABOT: We did several pelvic examinations and there was nothing.

A STUDENT: Was a lumbar puncture done?

DR. CABOT: Yes. It was normal.

A STUDENT: Was it a lesion in the lumbosacral region?

DR. CABOT: No evidence of any such thing in the X-ray.

I do not want to keep you in suspense. We ought to have thought of this thing. It is down in my book on differential diagnosis under pain in the back, and yet I did not think of it. After three weeks of this pain a herpetic eruption appeared. It was neuritis of the type of herpes zoster. The mistake was in neglecting to think of the possibility of that before the herpes appeared. We ought to have said "It may be that," and we ought to have maintained her courage, as we did not, against the terror of recurrent malignant disease. When that happens to you, remember the possibility of neuritis of the type that only later presents herpes on the surface. That is only one of the symptoms. Later she had loss of reflexes and atrophy; but at the time that we were so scared she did not have anything at all but terrific pain.

CASE 15152

THREE DAYS' COUGH WITH BLOOD-SPITTING

MEDICAL DEPARTMENT

An unmarried Canadian-American girl twenty-five years old, a student, entered December 24 complaining of cough for three days. She was so ill that the history was obtained from an aunt who was very ignorant of the patient's condition and very unintelligent.

Three days before admission the patient caught cold. She had malaise and aching in all the joints. She kept on working all the rest of the day. In the evening she went to a doctor, who said she had a cold and told her to go to bed. For the following day she continued about the same. Her aunt believed that she had fever. The night before admission she coughed up over a cupful of pure blood at one time. This was followed by a few mouthfuls of blood. After this she developed severe pain in the left side.

An aunt died of tuberculosis.

Clinical examination showed a poorly developed and nourished girl, very cyanotic, dyspneic and ill. The examination was not good on account of her condition. The skin was hot and dry. The mucous membranes were very cyanotic. The throat was injected. The lungs were full of moist bubbling râles, front and back. The lower right back showed dullness from the seventh to the tenth ribs. The heart borders could not be percussed. The apex impulse was not seen or felt. The sounds were not heard because of the loud râles. The pulse was thready and barely felt. The abdomen, extremities, pupils and knee-jerks were normal.

Temperature 103.8°. Pulse 130. Respirations 45.

Thirty minutes after admission the patient had a hemorrhage of bright red blood and died.

DISCUSSION

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

Whether or not she was exposed to tuberculosis from her aunt we do not know.

DIFFERENTIAL DIAGNOSIS

What are the possibilities? We certainly can say that she died of some disease of the respiratory tract or of the lung. Can it be *tuberculosis?* Yes. Is there anything against it? I believe not. How can you have it lasting only three days? I do not believe it lasted only three days. I shall have to go beyond the history and say it lasted longer, if it is tuberculosis.

What else could it be? It could be *pneumonia*, although the quantity of blood is unusual. Spitting up pure blood is not unusual in pneumonia, but spitting up this amount of blood is. I cannot exclude pneumonia.

Malignant disease of the lung cannot be excluded by any method that I know; but it is improbable if we accept the short history. One generally hears of malignant disease more than two days before death.

Lung abscess can of course give hemorrhage, but usually we hear a good deal more about

it and other symptoms like foul sputum earlier in the course of the disease.

Those four things are the only common ones that I know how to explain in such a picture as this. I think we can rule out malignant disease of the lung from the history. I think we can rule out abscess of the lung from the history. It comes down, then, to the question of tuberculosis and pneumonia. She is poorly nourished, and if we suppose that her cough has lasted more than three days I do not see anything against tuberculosis. Then comes the question of pneumonia. There is nothing said about a herpetic eruption such as we often see in pneumonia, but its absence is not against it. The sputum is perfectly consistent with influenza pneumonia. The amount of blood that comes up with that is larger than in other types of pneumonia. I see nothing to exclude influenzal pneumonia. It seems to me it comes down to those two. I favor the latter, *pneumonia, probably influenzal.* Tuberculosis cannot be excluded.

A STUDENT: Caseous pneumonia—what of that?

DR. CABOT: With that we ought to have more obvious signs of consolidation. The rapidly spreading râles and the blood do not sound like that to me.

A STUDENT: Tuberculous pneumonia—is that probable?

DR. CABOT: That is possible, yes.

A STUDENT: Could it be influenzal pneumonia on top of tuberculosis?

DR. CABOT: I suppose it could be, but I do not know how we could tell it.

A STUDENT: Did you think of typhoid fever?

DR. CABOT: We can rule it out. You do not die of typhoid (except with intestinal perforation or hemorrhage) unless you have been sick a long time.

A STUDENT: Is this consistent with pulmonary infarct?

DR. CABOT: Yes. But we have no suggestion as to where it might come from. I should not make a diagnosis of pulmonary infarct unless we have suspicions of trouble in the heart or peripheral veins from which the infarct can come.

A STUDENT: How about the joint symptoms?

DR. CABOT: I do not think we have much in the way of joint symptoms.

A STUDENT: Don't you think that she died of the hemorrhage rather than the disease?

DR. CABOT: No. I do not think she has lost enough blood to kill her.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Lobar pneumonia.
Tuberculous pneumonia?

DR. RICHARD C. CABOT'S DIAGNOSIS

Pneumonia, probably influenzal.
(Possibly tuberculosis.)

ANATOMIC DIAGNOSES

1. *Primary disease.*

Influenzal pneumonia.

2. *Secondary or terminal lesion.*

Necrosis of Malpighian corpuscles of spleen.

DR. TRACY B. MALLORY: This girl did have an influenza pneumonia. Both lungs were considerably distended, rather diffusely but not very firmly consolidated. On section they were deeply congested, hemorrhagic, very wet. The gross appearance was quite characteristic of the acute stage of the pneumonias that follow influenza. Cultures from the lungs showed approximately equal numbers of influenza bacilli and staphylococci. So far as I know the secondary invading organisms of these influenza pneumonias can vary considerably without any great difference in the clinical picture. A hemolytic streptococcus or pneumococcus is much more frequent than a staphylococcus. The latter is however by no means rare.

Everything else was entirely negative except for the microscopic finding of focal necroses in the spleen. They are to be regarded only as the anatomic evidence of a severe toxemia.

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PUBLIC HEALTH PROBLEMS BEFORE THE LEGISLATURE*

AT the opening of the Legislative Session the Governor outlined a program for the advancement of the public welfare. It met with a very favorable reception. A voluntary committee of citizens was formed to aid in carrying out the suggestions. A number of physicians offered their aid. Much of the program had to do with health matters.

It is well recognized that much more depends upon those of the medical profession who must be co-öperative in carrying out the recommendations than on the details of the program itself. The profession stands ready to serve.

Tuberculosis, cancer, mental disorders and prevention of the communicable diseases are the main problems of public health today. In tuberculosis tremendous progress has been made. In the campaign against cancer there is great promise of benefit through prompt detection of the early stages of the disease. The vast problems involved in mental diseases are being studied from many angles. The prevention of many of the communicable diseases depends on the adoption of well known methods which the medical profession has made available.

The states, counties, municipalities, hospitals and health organizations have invested millions of dollars in these vast and important undertakings. The success or failure depends in the final analysis on giving to the medical profession and their co-workers opportunities to function. Now at a time when great forward movements are under way it is proposed to admit to the general practice of medicine groups of individuals trained in courses of a few weeks' duration in chiropractic schools. The teaching of these schools flaunts all medical science and impedes medical progress.

Because many persons feel that they have been benefited by having their backs rubbed and have been led to believe that certain vertebrae have been adjusted it is proposed that chiropractors be granted special favors in the right to diagnose and treat general and local diseases, thus taking on the practice of medicine.

For years the medical profession has endeavored to raise the standards of medical education and practice. The purpose of these attempts has been to give better service to the public. But a cult seeking special favors threatens to lower the existing standards. The proposals are so bad that many physicians regard their accomplishment as unthinkable. They should note that the Committee on State Administration unanimously reported Senate Bill 304. The Editorial of the *Boston Herald* reprinted in a former issue referred to this bill. Members of the Legislature, who previously have favored sound standards of medical practice, are yielding to the various forces at work to gain special favors.

The attitude displayed by the Chairman of the Senate Committee on Ways and Means in the hearing upon the Chiropractic Bill, Senate 304, is a challenge to the entire medical profession of Massachusetts.

If the Governor and the Legislature are in any way in earnest in a welfare program, the time has come for plain statements. Those who have been chosen as public servants can no longer expect to grant special privileges to inadequately trained cultists and at the same time look for gains in the treatment and prevention of diseases which present such serious problems for the State.

History records the rise and decline of many cults. There is no evidence endorsed by qualified scientists that chiropractic has contributed anything of value in conditions associated with demonstrable pathology.

The medical profession, the health authorities, the governing boards of hospitals and the vast silent mass of our citizens depending upon able physicians and good hospitals must exert all possible influence to prevent this backward step.

*Attention is also called to the editorial on page 787, "Can This Be So?"

CHRISTMAS SEALS

THE success of the Christmas seal sale in raising money for the prosecution of tuberculosis work led the American Society for the Control of Cancer, Inc. to consider selling seals to help finance the work of the latter organization.

Dr. Kendall Emerson and Dr. Williams of the National Tuberculosis Association have met the Board of the American Society for the Control of Cancer, and an agreement that the latter named society will not engage in a seal sale this year has been reached.

This is fortunate because the adoption of this method of raising money by other organizations would seriously interfere with the popularity of this appeal. Since the present Christmas seal sale has been organized by the Tuberculosis Association, it is fair to leave the field to this body.

The Society for the Control of Cancer has a very definite appeal which can become effective under some other method. Although much money has been appropriated for the study of cancer, and able investigators are working on the subject, there is still opportunity for the expenditure of money in attempts to solve the mystery of the disease. The money is being supplied to a great extent by foundations and local organizations and progress is being made. If more money is needed, it will be forthcoming when sufficient publicity is given to the people.

DIAGNOSIS BY OPTICAL MACHINES

SPACE has been given in the newspapers to the alleged demonstration of disease by certain optical machines.

Ophthalmologists have long been able to detect changes in the retina and other structures of the eye which indicate definite physical changes throughout the body, but some of the claims for diagnosis by a machine are comparable with the theories of the departed Abrams. Even more brilliant illumination of the inner recesses of the eye will require interpretation by competent observers if errors are to be avoided.

While acknowledgment is cheerfully made of the debt of medicine to wonderful mechanical aids the public should not be led to believe that machines can do more than present evidence which requires expert interpretation.

As an illustration we know that even the clearest x-ray pictures are often misleading unless correctly interpreted.

A COMMITTEE ON RELATIONSHIPS OF NURSES AND PHYSICIANS

DIFFERENCES have arisen from time to time between nurses and physicians regarding the scope and function of the nurse in various nursing activities. As is almost always the case, these differences are due to a lack of understanding between the two parties. As nurses and physicians are working with the same end in view, it is gratifying to learn that the Massachusetts State Nurses Association has, through its Public Health Nursing Section, formed a Committee on Relationships with the Medical Profession.

Particular problems which have arisen so far deal chiefly with Public Health Nursing, Industrial Nursing, and the nurse in relation to the giving of anaesthetics. It is the hope of the nursing group that the District Medical Societies will form committees in various localities, to which problems arising in regard to relationship may be referred. It is hoped that this will bring about complete understanding and coöperative response from the physicians.

The Chairman of the Nurses' Committee is Miss Sophie C. Nelson, care of the John Hancock Mutual Life Insurance Company, 197 Clarendon street, Boston. She may be reached by telephone—Kenmore 5400.

This step taken by nurses and their request for aid and advice from physicians ought to meet with cordial help from the Massachusetts Medical Society.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

SEVER, JAMES WARREN. M.D. Harvard 1901. F.A.C.S. Assistant Orthopedic Surgeon, Children's Hospital, Boston. Instructor in Orthopedic Survey, Harvard Medical School. Orthopedic Surgeon, Cambridge Hospital, Cambridge; Waltham Hospital, Waltham; Massachusetts State Hospital School for Cripples, Canton; Industrial School for Crippled and Deformed Children, Boston; Symmes Arlington Hospital, Arlington. Consulting Orthopedic Surgeon, General Stephen Gale Hospital, Haverhill. Member of the American Orthopedic Association. His subject is: "Some End Results of Fractures About the Elbow Joints." Page 745. Address: 321 Dartmouth Street, Boston.

JESSAMAN, L. W. M.D. Tufts College Medical School, 1909. F.A.C.S. Oto-Laryngologist and Ophthalmologist, Framingham Union Hospital and Massachusetts Reformatory for Women. Visiting Ophthalmic Surgeon, Boston City Hospital. His subject is: "Treatment of Nasal Fractures." Page 753. Address: 132 Union Avenue, Framingham, Mass.

WATTS, JAMES, W. B.S., M.D. University of Virginia, 1928. Neuro-Surgical House Officer, Massachusetts General Hospital. Address: Massachusetts General Hospital, Boston. Associated with him is:

VIETS, HENRY R. B.S. M.D. Harvard, 1916. Instructor in Neurology, Harvard Medical School. Assistant Neurologist, Massachusetts General Hospital. Visiting Neurologist, Long Island Hospital. Their subject is: "Tuberculous Meningitis with an Unusual Cerebrospinal

Fluid." Page 757. Address: 6 Commonwealth Avenue, Boston.

LEARY, TIMOTHY. A.M. M.D. Harvard, 1895. Professor of Pathology and Bacteriology, Tufts College Medical School. Medical Examiner, Suffolk County. His subject is "Early American Experiences Under the Coroner Laws." Page 759. Address: 416 Huntington Avenue, Boston.

PAGE, IRVINE H. S.B. M.D. Cornell University Medical School, 1926. Assistant Visiting Physician, Presbyterian Hospital, New York and Kaiser Wilhelm Institute, Munich, Germany. Address: Kaiser Wilhelm Institute, Munich, Germany. Associated with him is:

WARREN, SHIELDS. A.B., M.D., Harvard, 1923. Pathologist, New England Deaconess Association Hospitals and New England Baptist Hospital. Director, State Diagnosis Service. Instructor, Harvard Medical School. Their subject is: "The Pathology of Diabetes in Young Adults." Page 766. Address: 195 Pilgrim road, Boston.

BLAISDELL, E. R. M.D. Bowdoin Medical College, 1915. Visiting Physician, Maine General Hospital, Maine Eye and Ear Infirmary, Portland City Dispensary. His subject is: "Sugar Tolerance and Hypertension in Cataract Patients." Page 768. Address: 12 Deering Street, Portland, Maine.

MORSE, JOHN LOVETT. A.M., A.B., M.D. Harvard Medical School, 1891. Professor of Pediatrics Emeritus, Harvard Medical School. Consulting Physician at Children's Hospital, Infants' Hospital, and Beth Israel Hospital. Member New England Pediatric Society, American Pediatric Society, Association of American Physicians and Boston Obstetrical Society. His subject is: "Progress in Pediatrics." Page 771. Address: 483 Beacon Street, Boston.

CAN THIS BE SO?

SINCE the editorial on Public Health Problems in this issue was written the *Boston Herald* has published comments on the situation surrounding the effort of the chiropractors to secure their special bill.

It is suspected that a powerful lobby is employed in the interest of this bill and it is feared that if it secures legislative endorsement, influences have been brought to bear which are not consistent with the highest ethical standards.

Those who are opposed to this legislation are not in favor of a paid lobby. They have appealed to the intelligence of the legislators. If any improper influence has been attempted in favor of this bill it is the duty of the legislature to institute a rigorous investigation. We urge everybody to read the Sunday *Herald* of April 7 and the Monday *Herald* of April 8. The suggestions therein contained are disquieting and the next move should be to ask for an investigation of the lobby employed to procure the passage of the bill for a special act to register chiropractors.

The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY

Frederick L. Good, M.D., *Chairman* **Louis E. Phaneuf, M.D.,** *Secretary*
Frederick J. Lynch, M.D., *Clerk*

What Are the Indications For (Operative) Delivery in the Second Stage?

Broadly speaking, the indications for operative delivery during the second stage of labor include all conditions which would jeopardize the life or health of the mother or child if labor is not speedily terminated. The recognition of these conditions requires close study of the general condition of the patient during pregnancy, a careful ante partum examination during the last month, and attention to the progress of labor from the standpoints of both mother and child.

It is essential, for the above purposes, that the possibility of passage of the baby through the maternal pelvis be accurately ascertained. If the physician is convinced of this either before labor, or after a test of labor which leads to full engagement of the vertex, it may be safely judged that delivery from below is feasible; and only if delivery from below is feasible does any indication exist for operative delivery from below. Thus, if the patient has an "absolute cesarian pelvis" with the true conjugate of 7.5 cm. or less, this point should have been recognized by appropriate ante partum examination.

Unfortunately a short discussion like this cannot do more than outline certain of the more common and important indications for delivery in the second stage. Much depends upon the judgment and skill of the physician himself, and many cases will occur which do not fit accurately into the classification below. However, we find that these indications may be maternal, fetal, or both, and a short analysis of some of the striking ones is here attempted.

1. MATERNAL.

a. Prolonged labor with maternal exhaustion, as shown by lapse of time, and the general symptoms and signs of fatigue on the part of the patient. This situation is probably the most common maternal indication. The first stage has been prolonged and characterized by nagging and inefficient uterine contractions. Frequently the baby presents in an occipito-posterior position, and frequently the membranes have been ruptured for some time. Maternal exhaustion is shown by a steadily rising pulse rate, lack of the normal tendency to bear down after the os is fully dilated, and in-

creasing irregularity and inefficiency of the uterine contractions, which may, indeed, cease entirely. Here the indication is for prompt delivery.

b. Valvular heart disease. The indication here is to relieve the patient of the physical stress incident to the second stage, thus avoiding expenditure of the cardiac reserve. This indication becomes imperative if the heart undergoes any degree of decompensation during labor.

c. Prolonged hypertension or threatened eclampsia. Here again the indication is to relieve the circulation of the burden imposed by the second stage, and to expedite emptying of the uterus.

d. Ante partum hemorrhage, which at this stage usually results from the premature separation of the normally implanted placenta. The uterus must be emptied to allow control of the hemorrhage.

e. Transverse presentation, compound presentation, persistent brow presentation, or face presentation with chin posterior, of the fetus. These conditions frequently lead to an impassé in the matter of spontaneous delivery, with resulting danger of maternal exhaustion and eventual rupture of the uterus.

2. FETAL indications for delivery during the second stage. Here we have, as the only early sign to guide us, changes in the rate and rhythm of the fetal heart sounds, which indicate interference with the fetal circulation and the danger or presence of asphyxia. A later sign, in prolonged or acute asphyxia, consists of the appearance of meconium from the vagina when, in the presence of ruptured membranes or on rupture of the amniotic sac, the baby is presenting by the vertex.

a. Prolonged labor, especially a prolonged second stage. Fetal distress is shown initially by a rapid heart rate, later slowing appreciably below 120 and becoming irregular.

b. Prolapse of the umbilical cord, when a long cord is forced down by uterine contractions, or is washed down by rupture of the membranes. Fetal death from asphyxia is practically certain unless the labor is terminated forthwith, or unless,—as is seldom feasible,—the cord can be replaced.

c. Presentation of the cord within the amniotic sac. This is often unrecognized until the membranes rupture or the second stage begins. A prolapsed cord is the inevitable result.

d. Other abnormalities of the cord, such as multiple turns around the baby's neck, entwining of the cord among the small parts, congenitally short cord, etc. These abnormalities can seldom be diagnosed ante partum but are often the cause of alarming and dangerous changes in the rate and rhythm of the fetal heart as the baby descends in the birth canal.

e. Premature separation of the normally attached or low attached placenta. Here the fetal circulation is rapidly jeopardized and the baby suffers asphyxia and death unless labor is quickly terminated.

f. Transverse presentations, compound presentations, persistent brow presentations, or face presentations with chin posterior. Here the mechanical imperfections of spontaneous delivery are such that the trauma and asphyxia which the baby is almost certain to suffer if the case is left to nature indicate operative delivery during the second stage.

Questions of a similar nature to the above will be discussed in the JOURNAL each week until June. They may be addressed to the Clerk of the Committee, in care of the JOURNAL and will be answered by members of the Committee of the Section of Obstetrics and Gynecology.

THE TREASURER'S REPORT

WITH RESPECT TO DIVIDEND DISTRIBUTION

THE Treasurer of the Massachusetts Medical Society makes the following report regarding the dividend distribution of April 2, 1929.

The Council of the Society voted to distribute the sum of $4000 to the District Societies this year. The total number of payments received in time to count for the dividend computation was 3438 making the dividend per each paid member $1.163467.

The following table gives the number of payments in each District and the dividend:

DIVIDEND FIGURING, APRIL 2, 1929
Total Reported, 3438
Total, $4000—per member, $1.163467

District	Number Reported Paid	Check
Barnstable	32	$37.25
Berkshire	88	102.39
Bristol North	51	59.33
Bristol South	176	204.77
Essex North	140	162.89
Essex South	169	196.63
Franklin	33	38.39
Hampden	232	269.92
Hampshire	54	62.83
Middlesex East	78	90.75
Middlesex North	105	122.16
Middlesex South	552	642.23
Norfolk	536	623.62
Norfolk South	73	84.93
Plymouth	87	101.22
Suffolk	664	772.54
Worcester	304	353.69
Worcester North	64	74.46
	3438	$4000.00
Non-Resident	203	
Total paid March 1, 1929	3641	

From March 1 to April 2 there have been 107 payments reported. This makes the total number of payments to date 3748.

Total paid March 1, 1928, 3713.

A. K. STONE, M.D., *Treasurer.*

THE BOSTON MEDICAL LIBRARY

TRENCH MOUTH

THE marked increase in the already prevalent disease of Vincent's Infection, commonly called "Trench Mouth", is such that it is attracting the widespread interest of both Medical and Dental Practitioners. This disease affects the soft tissues of the oral cavity and, unlike pyorrhea, is highly contagious. When neglected it is accompanied by a very rapid loss of the alveolar process, even to such an extent as to cause an entire loss of the teeth. It is first shown by bleeding gums, (which should always be a warning sign) followed by a grayish ulceration resulting in exposure of the roots and recession of the gums, opening up large holes in the interproximal spaces. In the early stages there is a fetid breath, which is typical, and when once observed can never be mistaken. In the acute stages there is considerable pain and frequently nervous irritability. The Spirochete of Vincent and the Fusiform Bacillus are the predominant organisms found upon microscopic examination, the numbers varying according to the stage of the disease.

The first step in every case of a suspected Vincent should be to take a smear from three different areas of the mouth. Stain and determine by microscopic examination the presence or absence of these two organisms, and if present, treatment should be immediately begun by using as a wash sodium perborate, a teaspoon to a glass of warm water every hour. This is followed by packing individual pledgets of cotton soaked in 15% Chromic Acid into each interproximal space, allowing each to remain a short period, followed by a rinse with sodium perborate. A convenient method of accomplishing this is by blocking off six teeth at a time, using cotton rolls and aseptic napkins, and when one area has been so treated, proceed to the next, covering both upper and lower jaws at one visit. This treatment should be repeated at two or three day intervals for two or three visits, using in the meantime the wash of sodium perborate.

When treatment has been carried to this point the acute symptoms have usually disappeared, pain has subsided and with comfort restored the patient feels that he is cured. The real situation is that each interproximal space that has been so treated is filled with dead tissues which will not regenerate, and unless careful curettement and removal of this tissue is carried out, the patient harbors a low grade infection which will bring on a recurrence of the disease every time resistance is lowered.

A careful curettement of the diseased soft tissues should be begun at this time and carried on until each interproximal space is thoroughly cleaned out. All overhanging fillings which could form a lodging place for bacteria should be made smooth, and at this point in the treatment should come the removal of any wisdom teeth which may retard a cure. The loose flaps of wisdom teeth, under which the organisms lodge, frequently prevent a complete cure of a Vincent's case and periodical recurrences continue until these teeth have been removed.

At this point also intra-oral trauma, if there is any present, should be corrected. We now should have a healthy oral condition established, but still the Vincent's treatment is not finished. Health restored must be maintained and this is accomplished by raising resistance. A few minutes period each day of massage of the gums properly done will in a short time change the entire tone of the tissue from a flabby condition to the hard, vigorous state of the healthy gum.

The fact that a Vincent's case has been cured does not give immunity from recurrence if the person is exposed to the same conditions that first caused it, but a Vincent's case once cured should never recur under any other conditions. The literature of "Trench Mouth" will be available in Holmes Hall during the fortnight beginning April 15.

CORRESPONDENCE

SUFFOLK DISTRICT MEDICAL SOCIETY

Office of the Secretary, 520 Commonwealth Avenue
Boston, April 6, 1929.

Editor NEW ENGLAND JOURNAL OF MEDICINE:

Owing to his illness, Dr. Walton Martin, of New York, will be unable to address the Suffolk District Medical Society on Wednesday evening, April 24.

We have been very fortunate in getting Dr. Henry K. Pancoast, Professor of Roentgenology at the University of Pennsylvania, to speak on "Radiation in Cancer". The discussion will be opened by Drs. Robert B. Greenough, Lawrie B. Morrison and George W. Holmes.

The subject of Dr. Pancoast's paper is of interest and importance to our profession. It is hoped there will be a large attendance at this meeting.

Sincerely yours,
ARTHUR H. CROSBIE, *Secretary.*

CRITICISM OF AN EDITORIAL

To the Editor,
NEW ENGLAND JOURNAL OF MEDICINE,
126 Mass. Ave., Boston.

Dear Sir:

I wish to say a few words regarding the remarkable editorial entitled "The Relation of Family Doctor and the Surgeon Specialist." Does the writer think that he is in Russia or Italy? In these United States every man graduates as a physician and surgeon and is recognized as such by all boards.

Many of the best men doing surgery in this country are or were in General Practice. Many surgeons have neglected Post-Operative Care—that is a well known fact. As for Anaesthetics, medical students, internes, and even nurses are allowed to administer to patients. In assisting at operations, the doctors

in the several states are certainly considered competent.

For years most of the doctors, probably 75%, have taken internships after graduation. In 1928 over 90% were taking internships in good hospitals. To say that the majority of them are not competent to assist, give ether, or take care of Post-Operative work is ridiculous. There are exceptions of course, such as Thyroid Cases, where it might be well to have special assistance.

Some three or four years ago it was stated by an A. M. A. Committee that 80% of all medical work could be done by the average General Practitioner and, from what I have seen and been able to find out, they are doing it in most of the States. In large cities, many men are not treating the bad cases that they should treat, but are sending them in to large hospitals where they are treated as "Cases", and in many instances getting very little care.

We are opposed to "Cults" and justly so. We are asked to help contribute to large Medical Libraries where only a few doctors, those living in the vicinity, will have the time to go and study, whereas, if Branch Libraries were established, more doctors could avail themselves of the privilege.

Advances have been made in medicine, but not to such an extent as those interested in a specialty would lead us to believe. "A house divided against itself shall not stand." The profession is certainly divided by the efforts of some to magnify beyond all reason the blessings of specialism. Representatives from certain Medical Societies go about telling lay trustees of Hospitals how they should be run and what doctors are fitted to practice therein, what work they should be allowed to do under supervision, the condition of their private patients noted and reported to the "chief." I did not know that Medical Ethics had been revised to allow a doctor to supervise the private patients of another.

The family doctor knows the patient and is especially interested in his welfare. He is the patient's trusted friend and his presence at the operating table has saved many a radical operation. In many cases he could do about as well as the surgeon, in others, his help and knowledge of the organs involved are certainly a benefit to the patient.

I agree with the writer that there are a few doctors not competent to give an anaesthetic, assist or take care of Post-Operative cases, but the great majority of American doctors are capable of doing so. If it is not true, then improved Medical Education in this country has been a failure.

It is known among medical men that efforts are being made to obtain all surgery for a few, the assisting as well.

Very truly yours,

FRANK E. ROWE, M.D.

RECENT DEATHS

THORNDIKE—DR. TOWNSEND WILLIAM THORNDIKE died April 5, 1929.

SHURTLEFF—DR. PERCY ANDREW SHURTLEFF, 53, died in Blandford, April 2, after an illness of about a year. He was born in Taunton, the son of Andrew G. and Emily (Briggs) Shurtleff. He had made his home in Blandford 25 years. Dr. Shurtleff formerly practiced in West Springfield.

SMITH—DR. WALTER ANSON SMITH, retired surgeon of Shelburne Falls, died at his home in that town, March 28, 1928, aged 72.

Dr. Smith was a graduate of the University of Vermont, College of Medicine in 1882. He joined the Massachusetts Medical Society from Cummington in 1884, later moving to Ashfield, Hinsdale and Springfield. In the last named city under the direction of the late Bishop T. D. Beaven he helped organize the House of Mercy Hospital in 1895 and served as surgeon to the hospital until his removal to Shelburne Falls in 1919. He was one of the founders of the Springfield Academy of Medicine. On account of failing health his name was placed on the retired list in 1922. Dr. Smith was a high degree Mason. His widow and a son survive him.

DUNN—DR. CHARLES STEIN DUNN, a Fellow of the Massachusetts Medical Society since 1894, died at his home at Haverhill after a long illness, January 7, 1929.

He was born at Dover, N. H., December 23, 1859, the son of George and Deborah Durgin Dunn. His education was obtained in the public schools of Dover and at Dartmouth College, though he did not take a degree. Entering Harvard Medical School he was graduated in the class of 1883 and settled in practice in Charlestown, Mass., in that year. In 1894 he moved to Haverhill, joined the state medical society and continued in practice until a few years before his death.

Dr. Dunn was survived by his widow, Mrs. Bertha Dodge Dunn, a daughter of the late Hervey Dodge of Newburyport, and by a son.

OBITUARIES

A TRIBUTE TO THE MEMORY OF JOSIAH CLARK HUBBARD

Josiah Clark Hubbard, M.D., died Monday, October 1st, 1928, at his home 321 Maple Street, Holyoke, Massachusetts, and Holyoke and this section of the state still mourn his passing.

He was born in Easthampton, Mass., September 5th, 1853. His father at that time was professor of Mathematics at Williston Academy there, and before and after this time held many teaching positions during his life, one of them being the first superintendent of the public schools of Springfield, Massachusetts. Dr. Hubbard's mother too was a teacher of much ability, and was one of the first graduating class at Mt. Holyoke Seminary, the second to none in its standing among the women's colleges of the country. Dr. Hubbard was therefore brought up among the surroundings of teachers and scholars, and this influence pervaded his whole life and living, for throughout his life to its end his library contained the best and latest of books and magazines and his conversations were well known for their richness of wisdom and these were ever remarked about because of their brilliancy and wit and because of the finest of quotations from his readings.

During his early childhood he was troubled with

an affection of his eyes which necessitated the wearing of glasses and prevented his reading, and corneal scars were the constant reminders of this early disability. For many years, and because of this eye trouble he had private tutors, who read aloud to him his lessons, and his mother for many years was accustomed to read to him for hours from the best literature available at this time. In this way he learned by heart many of the books of the Bible, and much of fine prose and poetical writings. I well remember how surprised I was, when in my early acquaintance with him, he began to recite, and quoted from his memory page after page of Scott's Lady of the Lake, and when he thought I had had enough, told me that he would give the rest any time I wanted it. This early eye trouble therefore was also, in its way, a great blessing, for the early memory never failed him, and the habit of memorizing was kept up into his last years.

Both parents being college graduates and teachers, his grammar school and high school training was therefore mostly of the best of home teaching, together with that of his private tutoring. Later on as his eyes improved he was able to do his own reading, beginning in his high school period, and continuing through his college and his professional school work.

His B. A. degree was from Williams College at Williamstown, Mass., where he graduated in the class of 1877. After leaving Williams College, his father wanted him to prepare himself for the life ot a teacher, for which the young man seemed well qualified. But this he did not seem to want to do. And rather against his father's wishes and advice, he went to Philadelphia, where under the influence of a physician and a friend of the family, who was a teacher at the medical school, he started the study of medicine. The very early capturing of scholarships and prizes soon quieted the objections of his father, and in the year of 1880 the son was graduated from the Jefferson Medical College, with honors. He spent one year after graduation in hospital work in Philadelphia, and then located for what turned out to be his life's work in Holyoke, Mass., in the year 1881, 47-48 years ago. Here he soon gained attention because of his ability and his scholarly bearing, and from those early days has been one of the leading physicians in this, his locality. And this place in the life of the community he has always held. And up until he retired from active practice in April 1928, his clientele was of the very best. The choicest of city practice was his, and many people who have been his clients for these 47 years, and who are now mourning his loss, were rather counting on his being their supervising and consulting physician friend, upon his return from abroad, although he was not to be in active practice again.

In his early life as a practitioner, he was always to be seen with the best of his profession, and took active part in the organizing and the building of the Holyoke Hospital. He was a member, a charter member, of the medical staff; was at one time the superintendent of the hospital; and when he gave up the active staff work, remained always as one of the valued members of its consulting staff. For many years he was a member of the executive committee of the staff, and was until his retirement. He was one of the advisory members of the trustees of the institution also, and during the last few years, when an outpatient department was being organized, he was chief of the medical staff in the department of internal medicine. Such were the many activities of his life, and all of his active work was kept up until March and April, 1928, when he retired from active practice entirely and after 47 years started abroad for a long period of rest and travel, which might ultimately have taken him around the world, but which unfortunately was interrupted by the onset of what turned out to be his last illness. If ever the saying is true, and it practically always is, that one can judge the character of a man, by the company he keeps, it was true of Doctor Hubbard. One never saw him or met him except on the highest level. The finest of our business men, of our medical men, of our teachers, and our preachers (many of whom from all walks of life were his clients) were to be seen with him always. His literary work he kept interested in and was a member of a select group of our local literary folk, in which group he presented many times the scientific side of life in such topics as "Hereditary Influences in Life, Co-ördinated with the Environmental".

As were his hospital associations, so were his medical society relationships. He was a member of the local, Holyoke Medical Society, and filled most of its offices. He was also a member of the Massachusetts Medical Society and the American Medical Associations, etc. In our local society, he was a member of the advisory committee, program committee, and ethics committee and the like, always filling a very useful position when anyone was needed.

Successful in being a "good doctor", although he did not amass great wealth, as he might have done with his ability in business, he was successful financially, and he had a fine home, filled with fine furnishings and paintings, and a fine library, and the pictures and works of art all within it. He was on the directorship of a number of our local banks, and business institutions in which he had made investments. He was very much a home man, and aside from his church association (Congregationalist) his medical societies and his literary work, spent his time at home in his library, with his family and friends. Aside from his school fraternity, he held no membership in secret or other societies. When he spent an evening out, it was to hear some noted lecturer, singer, musician, or groups as in symphony orchestras, etc. Otherwise he was at home with his family or friends.

His general appearance in every way coincided with what has been said of him above. His was the typical appearance of the gentleman and scholar. His early ill health, and the fact that he never was strong and rugged physically added to this general appearance. He was of average height, rather slim, yet straight. He carried his head up, and his eyes were ever alert. And as his hair changed to gray and then white in his later years, this general appearance never left him, nor did his quiet wit, or his other attributes, and during the last few weeks of his life and activities, as quoted by those who visited him, his manner was characteristic of the man he was—a gentleman and a scholar.

In April 1928, apparently in good health, he sailed for England, planning to travel as the spirit moved; later going on to the continent, and drifting along down to the Mediterranean sea and on. After enjoying very much some three months of this trip, an acute onset of what developed into his last illness turned him back toward home, which he reached in time to be among his friends and his

familiar surroundings at the end. Holyoke and this vicinity, his family and his friends will long remember him, a man very much worth while.

GEORGE D. HENDERSON.

SANFORD OSCAR BALDWIN

Sanford Oscar Baldwin died at his home in Framingham on March 24, 1929. Born in 1867 in Topsham, Maine, he graduated from Bowdoin College and in 1896 from Jefferson Medical College of Philadelphia.

After a period of post graduate study in Europe he settled in Framingham where he continued in active practice until his death, a period of thirty-one years.

He was always keenly interested in the Framingham Hospital and in the Training School for Nurses and gave devoted service to both these institutions. As physician to the State Normal School he guided wisely the health of pupils and faculty. He was a pioneer worker in another field of health activity. For a number of years he served as advisor to the executives of the Dennison Manufacturing Company. In this capacity he worked not only to guard them from sickness but to regulate their lives in such a manner as to give them the greatest possible well being and efficiency.

In private practice he was always a careful devoted skillful physician. The best interest of his patients was always his guiding motive. Beloved by those in all walks of life whom he had helped, he upheld always the highest ideals of medicine.

NEWS ITEMS

SMALLPOX AND VACCINATION—Three persons in a South Boston family are found to have smallpox. The disease was introduced by one of the family who is a traveling salesman covering territory in Maine and New Hampshire along the Canadian border.

None of the family were ever vaccinated.

We wonder if these people are among the objectors to vaccinations on grounds of religious belief. Perhaps they do believe in fire insurance if not in health insurance.

ILLNESS OF DR. WILFRED GRENFELL—Sir Wilfred Grenfell is reported to be ill in a Sanatorium with a strained heart and has been obliged to cancel his engagements. Lady Grenfell is quoted as saying that Dr. Grenfell's condition is not believed to be serious.

NOTICES

NEW ENGLAND HEALTH INSTITUTE

A School of Public Health known as the New England Health Institute is to be held in Hartford, April 22-26, 1929. Headquarters and all sessions will be at Hotel Bond, 320 Asylum Street, Hartford, Conn.

This is sponsored by the U. S. Public Health Service, Yale and Harvard Medical Schools, New England Colleges and State Departments of Health.

During the week of the Institute which begins at 2 P. M. Monday April 22, eighty-four lectures will be given by men and women well known in their respective fields of public health Administration, Preventable Diseases, Sanitary Engineering, Tuberculosis, Venereal Diseases, Child Hygiene, Public Health Nursing, Laboratory, Mental Hygiene, Foods and Food Control, Nutrition, Vital Statistics and Health Education.

On Tuesday evening there will be a banquet for the faculty and in honor of the fiftieth year of the Connecticut State Department of Health. Speakers at the Banquet will be Hon. John H. Trumbull, Governor of Connecticut, Dr. Edward K. Root, Dr. Hugh S. Cumming, Surgeon General, and Dr. George E. Vincent, President Rockfeller Foundation.

Preliminary Programs giving general information, schedule of hours for the different subjects and names of the speakers are now available. Copies of these will be mailed on request.

For further information address: New England Health Institute Headquarters, Connecticut; State Department of Health, 8 Washington Street, Hartford, Conn.

POST-GRADUATE COURSE AT THE UNIVERSITY OF BORDEAUX

There will be a Post-Graduate Course in Ear, Nose and Throat surgery for American Physicians at the University of Bordeaux, France, commencing July 22, 1929.

Dr. Leon Felderman, Philadelphia, Pennsylvania, is in charge of registering the American Physicians for this course.

THE LAWRENCE CANCER CLINIC

The regular Lawrence Cancer Clinic, to be held at Lawrence General Hospital, One Garden Street, Lawrence, upon Tuesday, Apr. 16 at 10 A. M., will be a Demonstration Clinic with Channing C. Simmons, M.D., of Boston, Associate in Surgery at Harvard University Medical School, and Surgeon to Collis C. Huntington Memorial Hospital, Boston, present as consultant. You are invited to accompany any of your patients whom you desire shall have this service, or to send them with a note, and a report will be returned to you. This service is gratis. Your attendance at the Clinic is always welcome.

Committee

ROY V. BAKETEL, M.D.,
CHAS. J. BURGESS, M.D.,
FRED'K D. MCALLISTER, M.D.,
JOHN J. MCARDLE, M.D.,
HARRY H. NEVERS, M.D.,
THOS. V. UNIAC, M.D.,
J. FORREST BURNHAM, M.D., *Chairman*.

REMOVAL

Dr. Samuel C. Gwynne announces the removal of his office to Rooms 206-207 Professional Building, 27 Elm Street, Worcester, Massachusetts.

NOTICES OF MEETINGS

CENSORS' MEETING

The Censors of the Suffolk District Medical Society will meet for the examination of candidates at the Medical Library, No. 8 The Fenway, Thursday, May 2, 1929 at 4.00 o'clock.

Candidates should make personal application to the Secretary, and present their medical diploma at least one week before the examination.

ARTHUR H. CROSBIE, *Secretary*.
520 Commonwealth Avenue, Boston.

BOSTON ORTHOPEDIC CLUB

There will be a meeting of the Boston Orthopedic Club in the Old Surgical Building, Amphitheatre, Boston City Hospital, on Monday, April 15, 1929, at 8:15 p. m.

PROGRAM

Frederic J. Cotton, M.D.
Classification of ankle fractures, and a new type.
Lime deficiency
Non-union intrauterine fracture
Spontaneous fracture both femoral necks
Spontaneous fracture (?) in adult
Two stage grafts to stumps and scars.

Otto J. Hermann, M.D.
Physiotherapy and apparatus in the convalescence of fractures—simple and compound.
Os calcis cases.

J. H. Shortell, M.D.
Carpal injuries.

J. H. Burnett, M. D.
Fracture surgical neck of the humerus.

Richard Berg, M.D.
Routine care of fractures of humeral shaft.

R. K. GHORMLEY, *Secretary.*

THE ANNUAL MEETING OF THE MIDDLESEX SOUTH DISTRICT MEDICAL SOCIETY

The Middlesex South District Medical Society will hold its annual meeting on Wednesday, April 17, at the Commander Hotel, Cambridge.

Business meeting at 11:30 A. M.

The Annual Oration will be delivered by Dr. John Lovett Morse at 12 o'clock noon. Subject: "The Problem of the Control of Infantile Paralysis."

The Annual Dinner will be served at 1 P. M.

DR. A. W. DUDLEY, *Pres.*
DR. S. M. BIDDLE, *Sec.*

BOSTON MEDICAL HISTORY CLUB

ANNUAL MEETING

Monday, April 15, 1929, at 8.15 P. M. Boston Medical Library.

Remarks by the President.
Reports. Election of Officers.
A layman's appreciation of William James. R. W. Coues, Esq.
Some medical aspects of Burton's "Anatomy of Melancholy". Isador H. Coriat, M.D.
Haarlem Oil. James F. Ballard, Esq.

JAMES F. BALLARD, *Secretary.*

THE CLINICAL CONFERENCE OF THE NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The monthly clinical conference of the New England Hospital for Women and Children will be held at the Hospital, Dimock Street, Roxbury, on Thursday, April 18, at 8 P. M.

The following cases will be presented with special reference to their pathological findings; Internal Hydrocephalus and Cerebral Hemorrhage, Congenital Anomaly of the Duodenum, Puerperal Septicemia, Lymphatic Leukemia, Carcinomatosis.

ALICE H. BIGELOW, *Secretary.*

THE BETH ISRAEL HOSPITAL

Beth Israel Hospital
330 Brookline Avenue
Boston, Mass.
Charles F. Wilinsky, M.D. Director

April 3, 1929.

There will be a Meeting at the Beth Israel Hospital, April 22, 1929. Dr. Edgar Mayer of Saranac Lake, New York, will read the paper entitled "The Use of Pneumothorax, Phrenicotomy and Thoracoplasty in the Treatment of Pulmonary Tuberculosis." Dr. John B. Hawes II and Dr. Harlan Newton will open the discussion. There will be a presentation of cases.

HERRMAN L. BLUMGART, M.D.

COMBINED MEETING OF THE NEW ENGLAND PEDIATRIC SOCIETY AND THE CERTIFIED MILK PRODUCERS' ASSOCIATION

A combined meeting of the New England Pediatric Society and the Certified Milk Producers' Association of Metropolitan Boston will be held at the Boston Medical Library, 8 The Fenway, on Friday, April 12, 1929, at 8:15 P. M.

The following papers will be presented:

1. The Present Standards of Certified Milk in Boston, Richard S. Eustis, M.D., Secretary of the Medical Milk Commission of Boston, Inc.

2. Recent Advances in the Chemistry of Nutrition, H. C. Sherman, M.D., Professor of Food Chemistry, Columbia University, New York.

Light refreshments will be served after the meeting.

EDWIN T. WYMAN, M.D., *President.*
RANDOLPH K. BYERS, M.D., *Secretary.*

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

The next meeting of the New England Ophthalmological Society will be held at the Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston, on Tuesday evening, April 16, 1929, at 8 o'clock.

Clinical cases will be shown at 7.45 p. m.

PROGRAM

1. Post-operative Bleeding Following Chalazion Operation, Dr. Rowland

2. Juvenile Glaucoma, Dr. Riemer.

3. Cilium in Anterior Chamber 13 years, Dr. Riemer.

4. Intraocular Foreign Body in Vitreous for 1 year, Dr. Regan.

5. Anaphylactic Shock from Use of Foreign Protein, Dr. Liebman.

6. Unusual Case of Chorio-retinitis, Dr. Skirball.

7. Unusual Muscle Reactions, Dr. Dunphy.

8. Keratoglobus, Dr. Terry.

PAPER

Heterochromia Iridis with Cyclitis and Cataract, Dr. Harry Gradle of Chicago.

E. B. DUNPHY, *Secretary.*

Members reporting cases or reading papers are expected to have an abstract with them to hand to the Secretary.

NEW ENGLAND HEART ASSOCIATION

A joint meeting of the Hampden District Medical Society and the New England Heart Association will be held in the Supper Room of the Kimball Hotel, Springfield, Mass., on Tuesday, Apr. 16, 1929, at 4 P. M.

1. Regular business and election of officers for the ensuing year.

2. Cardiac Adequacy for Major Surgical or Obstetrical Procedures. Dr. Samuel A. Levine, Boston.

Discussion by Dr. J. M. Birnie and Dr. F. B. Sweet.

3 Coronary Artery Disease. Dr. William H. Robey, Boston.

Discussion by Dr. E. A. Bates and Dr. G. L. Steele.

SOCIETY MEETINGS

Combined Meeting of the Boston Medical Library, the Suffolk District Medical Society, the Middlesex South District Medical Society and the Norfolk District Medical Society (at Boston Medical Library at 8:15)

April 24, 1929—Annual meeting. Speaker, Dr. Henry K. Pancoast, Professor of Rontgenology at the University of Pennsylvania. His subject will be "Radiation in Cancer."

The medical profession is cordially invited to attend this meeting.

April 12—Combined meeting of the New England Pediatric Society and the Certified Milk Producers' Association. Complete notice appears on page 793.

April 15—Boston Medical History Club. Detailed notice appears on page 793.

April 15—Boston Orthopedic Club. Complete notice appears on page 793.

April 16—New England Heart Association. Detailed notice appears elsewhere on this page.

April 16—New England Ophthalmological Society. Detailed notice appears on page 793.

April 18—The Clinical Conference of the New England Hospital for Women and Children. Detailed notice appears on page 793.

April 22—Beth Israel Hospital. Detailed notice appears on page 793.

May 5-10—First International Congress on Mental Hygiene. Complete notice appears on page 411, issue of February 21.

June 13—International Hospital Congress. Complete notice appears on page 212, issue of January 24.

July 9—American Heart Association. Scientific Meeting from 2 to 6 P. M. in the Auditorium of the Woman's Club, 448 Taylor Street, between 12th and 13th Streets, Portland, Oregon.

October 7-19—New York Academy of Medicine. Detailed notice appears on page 468, issue of February 28.

DISTRICT MEDICAL SOCIETIES

Barnstable District Medical Society

Meeting: May 2, 1929.

Bristol North District Medical Society

April 18, 1929—Spring meeting.

Essex North District Medical Society

May 1, 1929 (Wednesday)—Annual meeting at Lawrence General Hospital, One Garden Street, Lawrence, at 12:30 P. M. (Telephone 5156.)

May 2, 1929 (Thursday)—Censors meet at Hotel Bartlett, 95 Main Street, Haverhill, at 2 P. M. (Tel. 3430.) Candidates should present their diplomas one week in advance to the Secretary, J. Forrest Burnham, M.D., 567 Haverhill Street, Lawrence.

Essex South District Medical Society

Thursday, May 2, 1929—Censors meet at Salem Hospital, 3:30 P. M. for examination of candidates. Candidates should apply to the Secretary, (Dr. R. E. Stone, Beverly) at least one week prior.

Tuesday, May 14, 1929—Annual Meeting. The Tavern, Gloucester. Speaker: Dr. O. F. Carr, Beverly, Trip around World, Illustrated. Ladies invited. Dancing.

Franklin District Medical Society

Meeting will be held on the second Tuesday of May.

Middlesex East District Medical Society

The following schedule has been arranged:

May—Melrose.

Subjects and speakers to be arranged by committee in each city or town.

Middlesex North District Medical Society

The annual meeting will be held the last Wednesday in April.

Middlesex South District Medical Society

April 17—Annual meeting. Detailed notice appears on page 793.

Norfolk District Medical Society

May—Annual meeting program to be announced.

The Censors of the Society meet May 9, 1929, at the Roxbury Masonic Temple, Warren Street, Roxbury, at 4 P M., for the examination of candidates.

Plymouth District Medical Society

April meeting at the Commercial Club, Brockton, April 18th, at 6 P. M. Annual Oration by Dr. Ralph C. McLeod, Orator; dinner.

Suffolk District Medical Society

See Combined Meeting in previous column.

May 2—Censors' meeting. Detailed notice appears on page 792.

Worcester District Medical Society

May 8, 1929—Annual meeting. Worcester Country Club

Worcester North District Medical Society

Annual meeting the fourth Tuesday in April.

BOOK REVIEWS

You—And The Doctor. By JOHN B. HAWES, 2ND, M.D. Houghton Mifflin Company, Boston and New York, 1929, xv + 181 pages. Price, $2.00.

One of the modern trends of medicine is towards a closer co-öperation between physicians and patients. The good results of the tuberculosis campaign and, more recently, the attack on cancer and heart disease, illustrate what can be accomplished by intelligent instruction of the public by qualified physicians. The author of this book enthusiastically supports this viewpoint; "education, more and more education of the laity in health matters, along with a better spirit of co-öperation between the public and the medical profession, is still needed. Hence this book".

Twenty-nine short chapters cover topics as diverse as "Headache", "Hemorrhage", "Nerves" and "The Family Medicine Closet". Frankness and honesty of purpose are evident throughout. Dr. Hawes says: "I dislike camouflage and there has been too much camouflage in medicine". He lets go what he terms "the mystery, the sacred rites and ceremonies" of medicine and talks of "health and how to keep well" in language easily understood by any intelligent reader.

There is little, in one sense of the word, to criticize; no "state secrets" of medicine, if there are any, are given away. The author's style is brisk, but at times rough and even harsh. Expressions like "hopeless abdominal tumors" grate on the ear; the book is replete with terms such as "eminent physicians", "authoritative opinions", "one of the greatest physicians and humanists", etc. Local pride at times prevents critical judgment. The main cri-

ticism of the book, moreover, is along the same line; the writer's point of view is distinctly "local" and superficial. Although his intentions are good, the subject is a most difficult one, and one feels a lack of ability on his part always to evaluate the importance of the material handled and to present it, as it should be presented, to the public. Books dealing in do's and don't's and generalities in medicine have always been difficult to write; this book by Dr. Hawes is better than most.

International Clinics. Volume IV. Thirty-Eighth Series, 1928. J. B. Lippincott Company.

This volume begins with a discussion of aging and old age, a subject which is attracting attention among medical men. Putti of Italy discusses arthritis deformans of the hip; Harlow Brooks presents certain phases of angina pectoris; Thomas Brown reviews the digestive problems of old age and Linsley Williams analyzes preventive measures.

In the realm of diagnosis and treatment we find an excellent X-ray study of the appendix and its significance is discussed by Bird. The progression of physical signs of appendicitis are diagrammatically represented by Behan. Solomon Solis-Cohen makes a plea for investigation of some of the older drugs found empirically valuable and urges their retention in the U. S. P. Pfahler presents encouraging results following the treatment of some malignant conditions by radiotherapy.

The outstanding feature of the surgical section is the excellent article by Haymann of Munich who evaluates the proper place and limitations of ligation of the jugular vein and compares statistically the ligated versus the non-ligated cases. As a result of his studies he lays down definite indications for the operation.

As usual the medical history and questionnaire portions are very interesting and the biographical sketch of Friedrich Kraus absorbing. There are many other excellent articles in this volume with illustrations of equal value. There are an unusually large number of typographical errors in this issue. In general the noteworthy character of the International Clinics reflects the high calibre of the editorial staff and is well worthy of perusal.

A Textbook of Medicine By Various Authors, Edited by J. J. Conybeare. New York, Wm. Wood and Company, 1929. Pp. 976. $8.00.

Whether another textbook of medicine is needed at the present time is extremely doubtful. With the many Systems of Medicine now published, and with the incomparable Osler-McCrae textbook, as standards, it would seem that there would be little room or demand for more works of this sort. The editor feels, however, that there is a place for a less bulky and lower priced volume which retains the character of a textbook and does not have the abbreviated form of a synopsis. The latest edition of Osler contains 1250 pages, the present volume 976. The claim for less bulk is justified; but as to price, Osler is somewhat cheaper.

It seems, then, that in order to have a less weighty volume, one must pay the price in sketchier articles, and this is exactly what has happened. American students will note the absence of anything on Tularemia, but this is to be expected for it is purely an American disease, although an increasingly important one to recognize. Other sections seem too abbreviated to make the book a useful one for reference. On the other hand, what there is of each section is, as a rule, well written and concise, particularly the sections on Visceroptosis. The section devoted to Common Diseases of the Skin is too brief to be of value, and as it lacks illustrations, it could scarcely teach anything to the inexperienced student. It is possible that this book may become a handy volume, but it will always require supplementation by a more complete text.

The Harvey Lectures, 1927-1928. Series XXIII. Williams and Wilkins, Baltimore, 1929, pp. 280.

This, the twenty-third series of lectures delivered under the auspices of the Harvey Society of New York, contains papers read by outstanding figures in medical investigation. The contents of the volume are as follows: "A Summary of Present Knowledge of Tularaemia", by Edward Francis; "The Relation of the Liver to Metabolism", by Frank C. Mann; "The Influence of Insulin and Epinephrine on the Fate of Sugar in the Animal Body", by Carl F. Cori; "The Metabolism of Nerves", by Wallace O. Fenn; "The Treatment of Pernicious Anemia", by George R. Minot; "Experimental Fistulae of Blood Vessels", by E. S. London; "The Renal Lesion in Bright's Disease", by Thomas Addis; and "The Development of the Harveian Circulation: William Harvey Tercentenary Address", by Alfred E. Cohn.

Although the subject matter contained in these lectures may have already appeared in medical literature and be more or less known to the medical public, the fact that it is scattered makes these lectures all the more attractive. Dr. Francis's paper by itself would justify one in buying the volume.

Tumors Arising from the Blood Vessels of the Brain. By Harvey Cushing and Percival Bailey. Charles C. Thomas, Springfield, Illinois, and Baltimore, Md. 1928. Price $7.50.

This volume is a very complete monograph on the blood vessel tumors of the brain. As one would expect in such a paper, each case is reported in detail and the discussion, particularly in regard to the pathological findings, is very extensive. The literature concerning these growths has been carefully searched and analyzed.

The first portion of the book is given over to the angiomatous malformations of the blood vessels of the brain and some fourteen cases are reported. The second portion deals with true new growths arising from angioblastic elements with detailed reports of eleven cases.

The section on symptomatology and treatment are condensed into a comparatively small number of pages. This seems rather a pity for one would like to learn in more detail the conclusions reached by the authors in regard to these two important subjects. The careful reader, however, will get much added information in this regard by detailed study of the case reports. Tumors such as these are so rare that it is only possible to assemble such a group in the wealth of material brought together in Dr. Cushing's clinic.

Such a study as this is not a text book for medical students or even for the reading of the more

studious practitioner of medicine. It will be of great value to that growing group of medical men whose footsteps follow the more recently opened trails in neurology, neuropathology and neurosurgery,

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Angina Pectoris. By HARLOW BROOKS, M.D. Harper Brothers, New York, 1929. Price $2.50.

This pocket edition gives a fairly complete picture of angina pectoris. The entire point of view taken by the author is a practical one, which makes it very readable. It is not meant particularly for experts, but rather for the general practitioner and it might even be profitable to the patients who suffer from this disease. Considerable attention is given to treatment which is a very welcome feature in any modern medical book. Numerous references from personal experience and illustrative cases help greatly in making this treatise enjoyable and clear.

As to the substance contained in the discussions, there is very little to criticise and much to commend. It is somewhat surprising, however, to read on page 29, that angina pectoris is considered to be unassociated with hypertension during the immediate attack—in fact the author states that blood pressure is commonly lower than the customary pressure of the individual at this time. There is much available data indicating the reverse of this view. Speaking of the factors which are most likely to induce attacks of angina pectoris, the author states that "the physician will do well to listen to the prejudices of his patients in this respect, for the range of idiosyncrasies is large and extremely varied." This is very sound advice and in fact typifies the entire attitude that the physician should take towards the patient in studying this disease, for the history and the patient's individual experiences are much more important than the physical examination. Altogether this book makes an excellent simple summary of this very common disease.

Spinal Anesthesia. By CHARLES H. EVANS, M.D., Clinical Assistant, New York Post-Graduate Medical School and Hospital; Lying-In Hospital of the City of New York; Assistant Surgeon, New Jersey Orthopedic Hospital, Orange. Published by Paul B. Hoeber, Inc., New York. January, 1929. 203 pages. Price $5.50.

In his monograph on Spinal Anesthesia Evans takes up the subject from two angles. He first discusses the physiology which is involved in the production of this form of anesthesia, and follows this theoretical aspect of the subject by the description, in detail, of the actual method which he has found to be most satisfactory. His discussion of the theory of spinal anesthesia and its complications is the most thorough which we have seen. His method of inducing anesthesia appears to be based upon considerable experience, and is simple, cautious and adequate. For the anesthetizing agent he employs crystals of neocaine, which he dissolves in spinal fluid, and to maintain the blood pressure he uses intramuscular injections of adrenalin. He seems to be opposed to the employment of ephedrin for this purpose, although the experience of others has shown it to be a most helpful aid in maintaining the blood pressure.

Evans differs with Labat as to the wisdom of placing the patient in Trendelenburg position after the spinal injection has been made. The former advises against this unless cerebral anemia, due to lowered blood pressure, has occurred. Labat, if we remember correctly, prefers to have the patient's head lowered. As many operations are made much easier by tilting the patient, it would seem that Evans's idea on this phase of the question is wrong or, if correct, constitutes an argument against the method which he favors.

His book is to be recommended to those who wish to learn the fundamentals of this much exploited method of producing anesthesia. There is considerable repetition in it, particularly in the last few chapters, but perhaps this is not a fault. If the novice will follow Evans's direction conscientiously he will not go far wrong.

Surgical Clinics of North America, February, 1929, Vol. 9, No. 1. W. B. Saunders Co., Philadelphia.

This is the Mayo Clinic number, and as is true of most publications emanating from that source, it contains a wealth of valuable material.

Judd opens with a note of optimism by reporting two cases of very *extensive carcinoma of the stomach* invading the pancreas and in which cures were obtained. He then reports a case of *hemangioma of the duodenum* with a discussion of hematemesis, a case where a large number of stones were removed from the *common duct* ten years after a cholecystectomy, and a case of a small papillary squamous cell *epithelioma of the renal pelvis.* With Passalacqua he gives a very encouraging report on the use of *oxygen as a prophylaxis against pulmonary complications.*

Balfour contributes a clinic on gastric surgery dealing chiefly with *recurring peptic ulcer following partial gastrectomy.* Apparently even radical gastrectomy is no guarantee that gastro-jejunal ulcer will not occur.

Henderson and Meyerding present the orthopedic cases, the former discussing fractures of the hip and the latter presenting various cases and emphasizing the comparative frequency of traumatic spondylolisthesis. In every severe back injury lateral as well as anteroposterior roentgenograms should be taken.

From the laboratories of Mann come interesting discussions of the *significance of urea* and of the *phagocytic cells of the liver.* This power of phagocytosis is apparently limited to the von Kupffer cells.

Rankin presents varied cases with *tumors* in the *submaxillary* region and then discusses *sigmoid polyps* and the technic of transperitoneal sigmoidotomy.

Lundy reviews the use of the various *anaesthesias* at the Mayo Clinic.

One of the most interesting contributions is by Walters on *obstruction of the upper portion of the urinary tract.* He has had very satisfactory results following plastic operations on the kidney pelvis for hydronephrosis. He emphasizes the seriousness of obstruction in the presence of infection.

Craig presents a very able account of *spina bifida.* He advocates delaying operation to the age of nine months using care to prevent abrasion or infection in the meantime.

A comprehensive discussion of *anal fissure* and its treatment by a thorough operation is given by Buie.

Many equally valuable clinics are presented by Sistrunk, New, Harrington, Pemberton, Hunt, Bumpus and others which are sure to be of interest to the surgeon.